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FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

OFFICIAL ORGAN OF
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CHARLES C THOMAS · PUBLISHER · SPRINGFIELD · ILLINOIS

VOLUME 86 JULY, 1961 NUMBER 1

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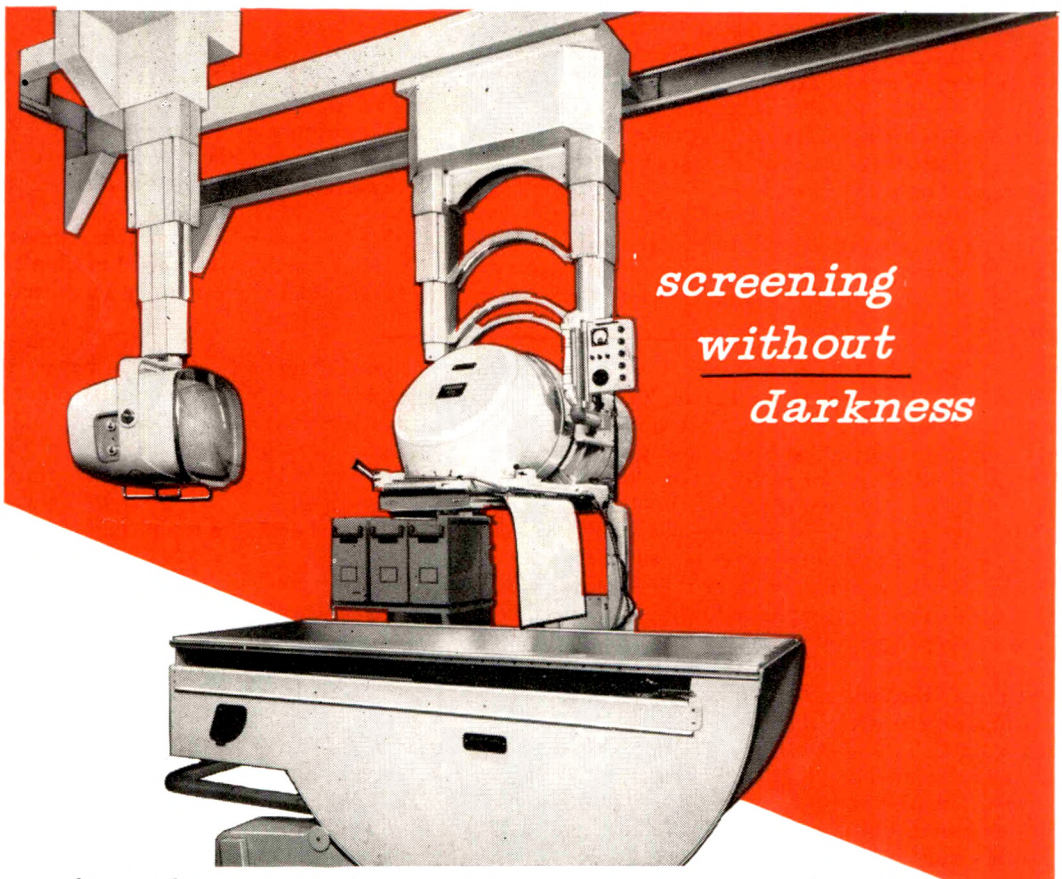
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Entered as Second-class at Springfield, Illinois under the
Act of March 3, 1879. Printed in U.S.A.

Second-class postage paid at Springfield, Illinois
and at additional mailing offices.

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[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

The AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

Editor: LAWRENCE REYNOLDS, M.D.

Assistant to the Editor: KENNETH L. KRABBENHOFT, M.D.

VOLUME 86

JULY–DECEMBER, 1961

CHARLES C THOMAS : SPRINGFIELD, ILLINOIS
1961

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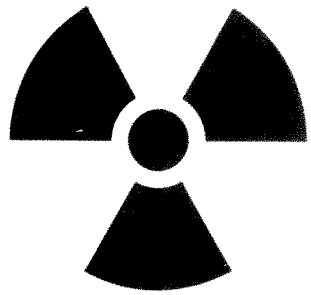
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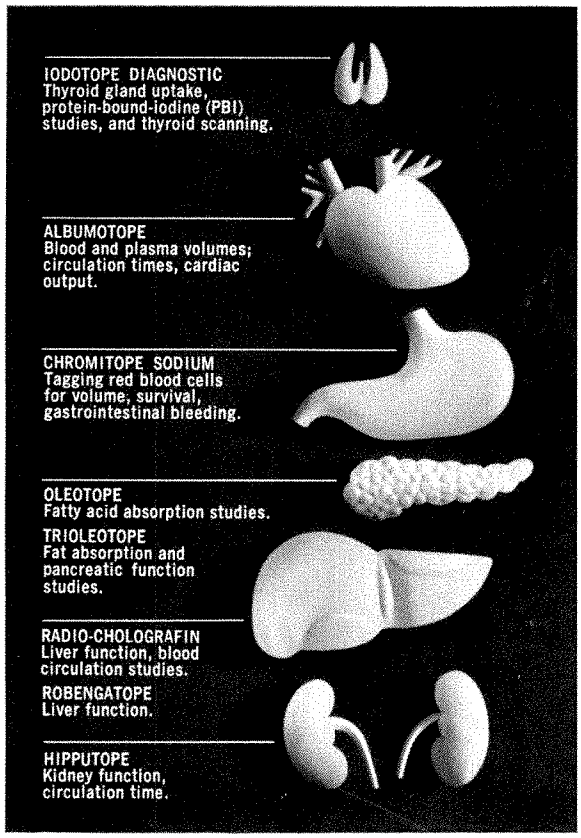
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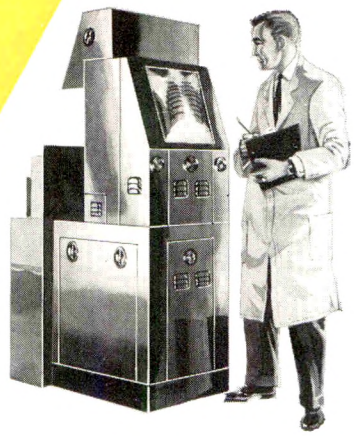
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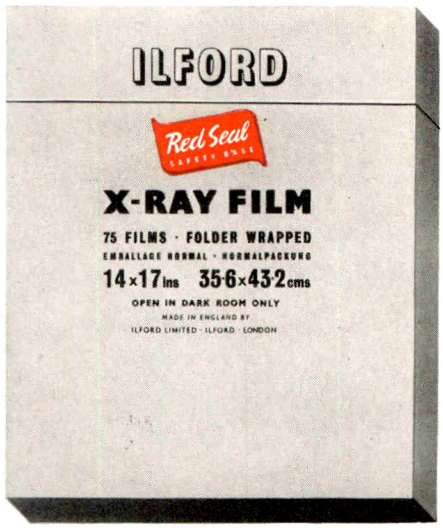
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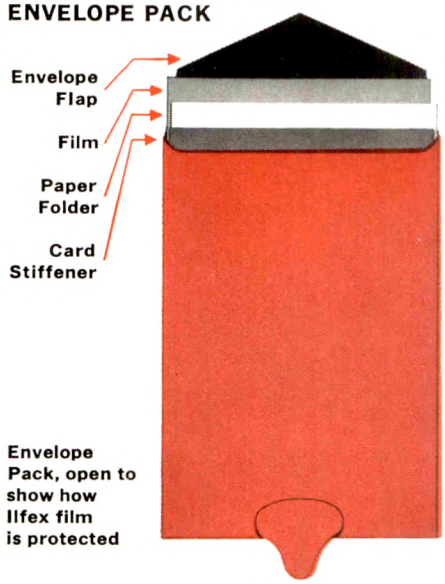


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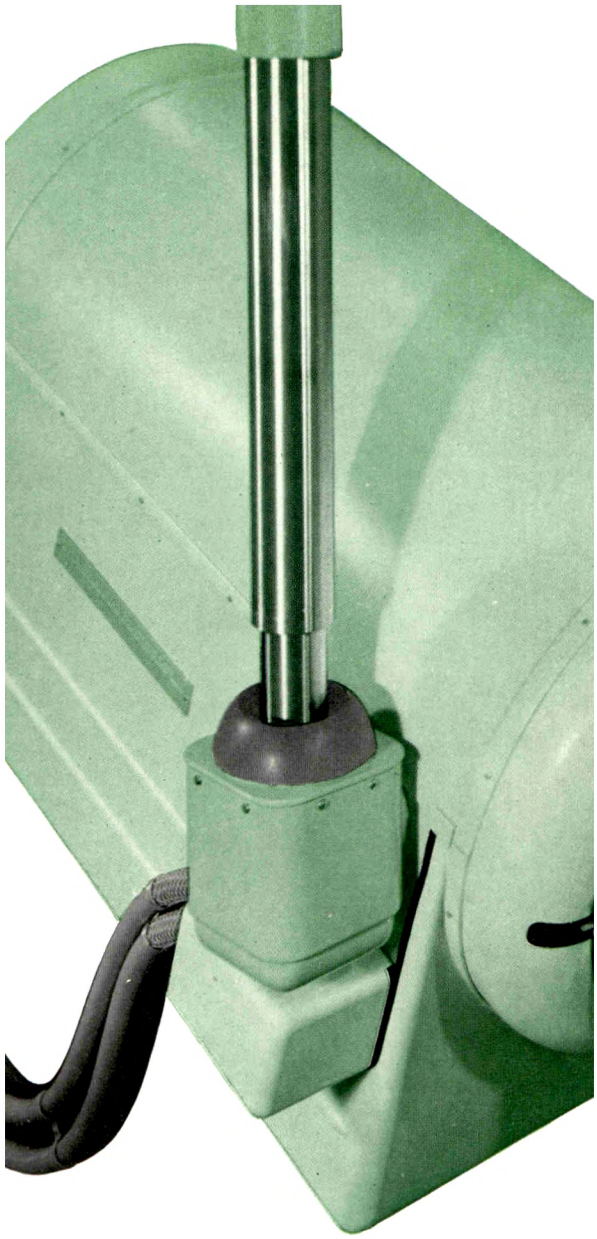
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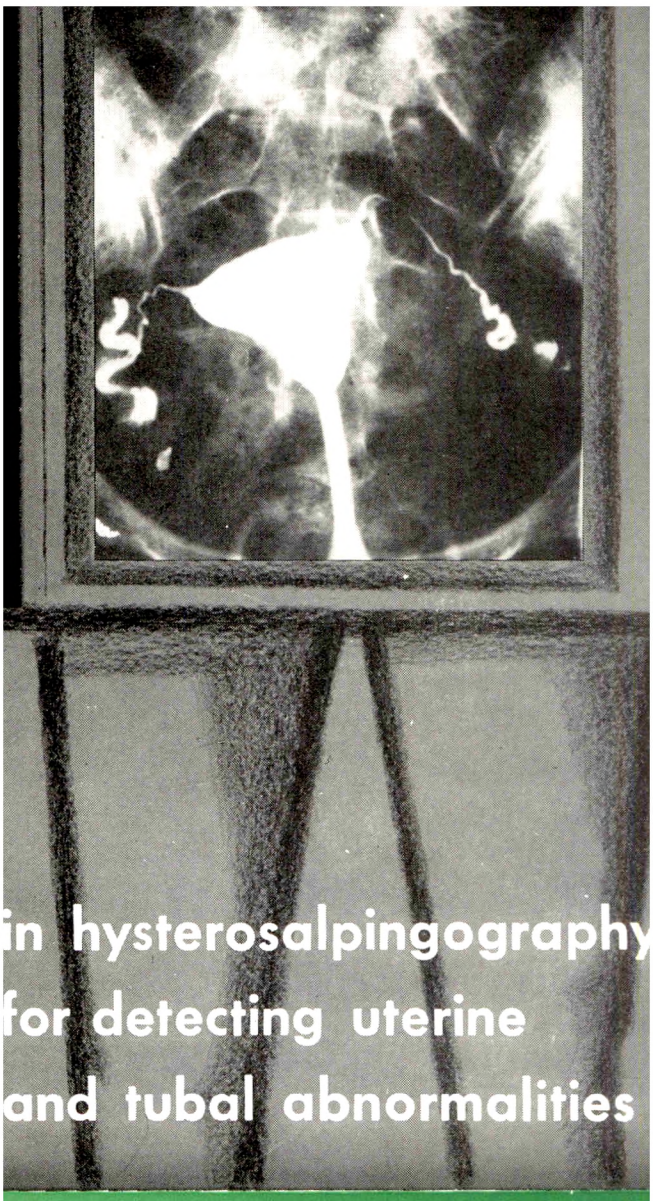
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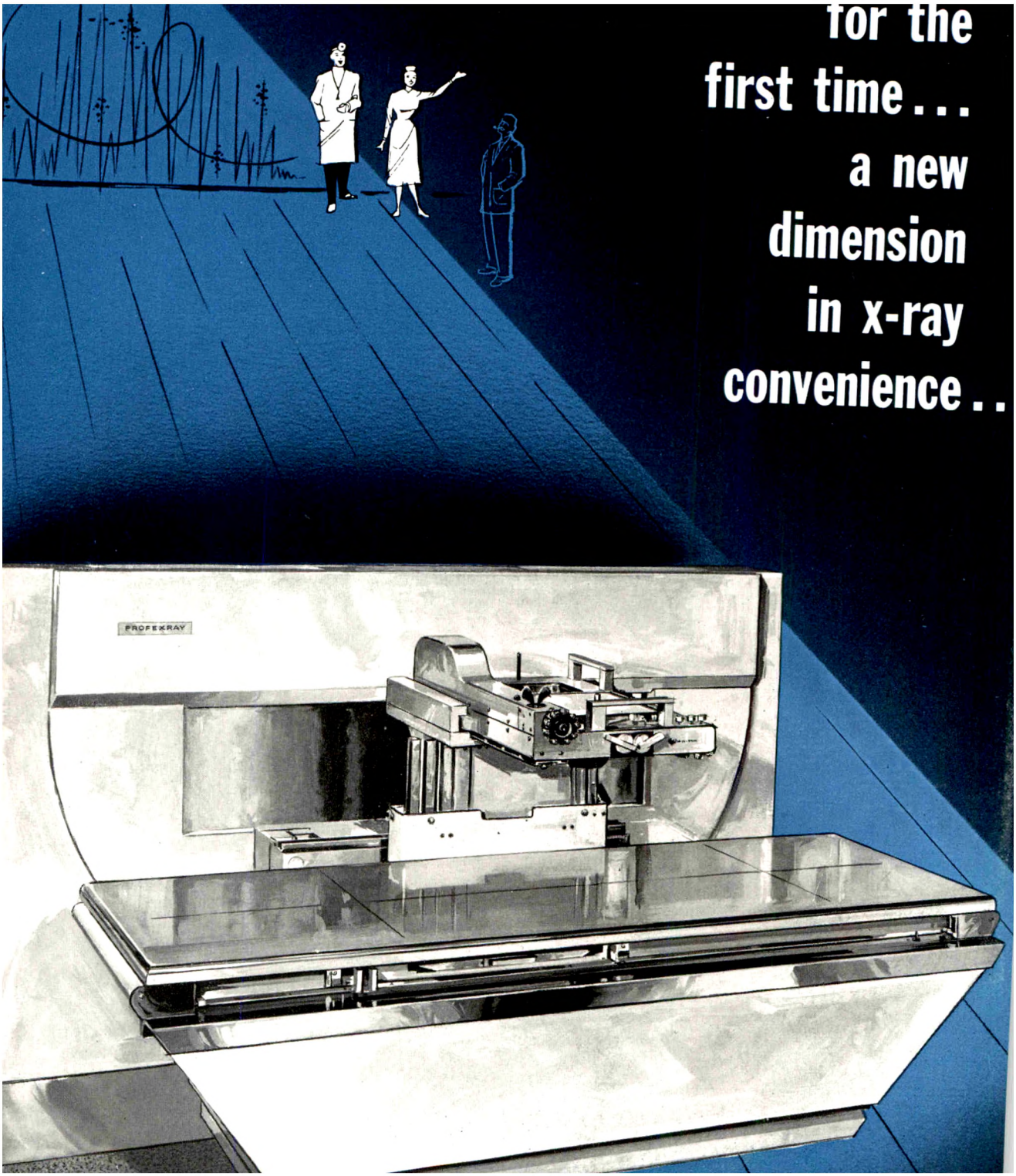

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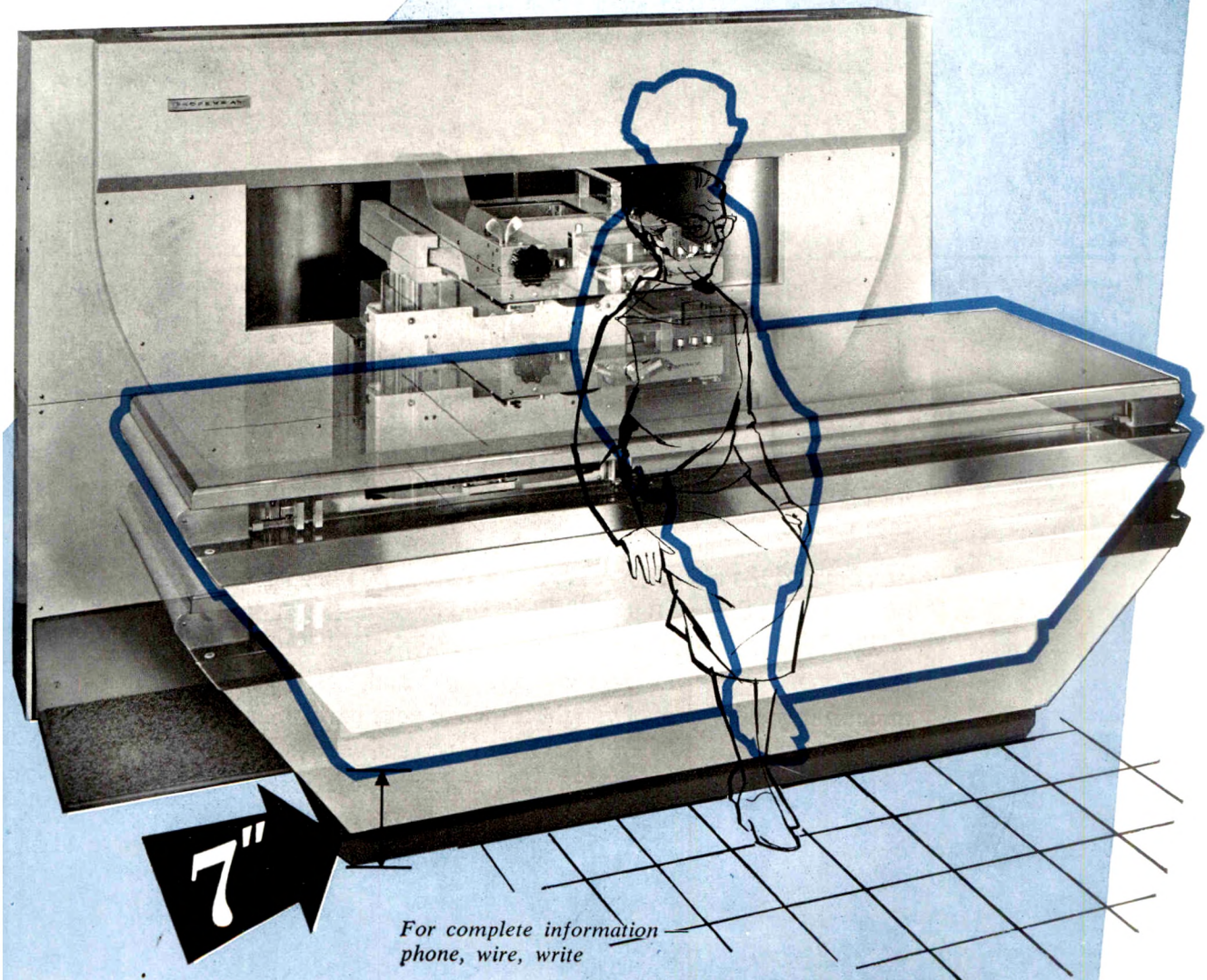
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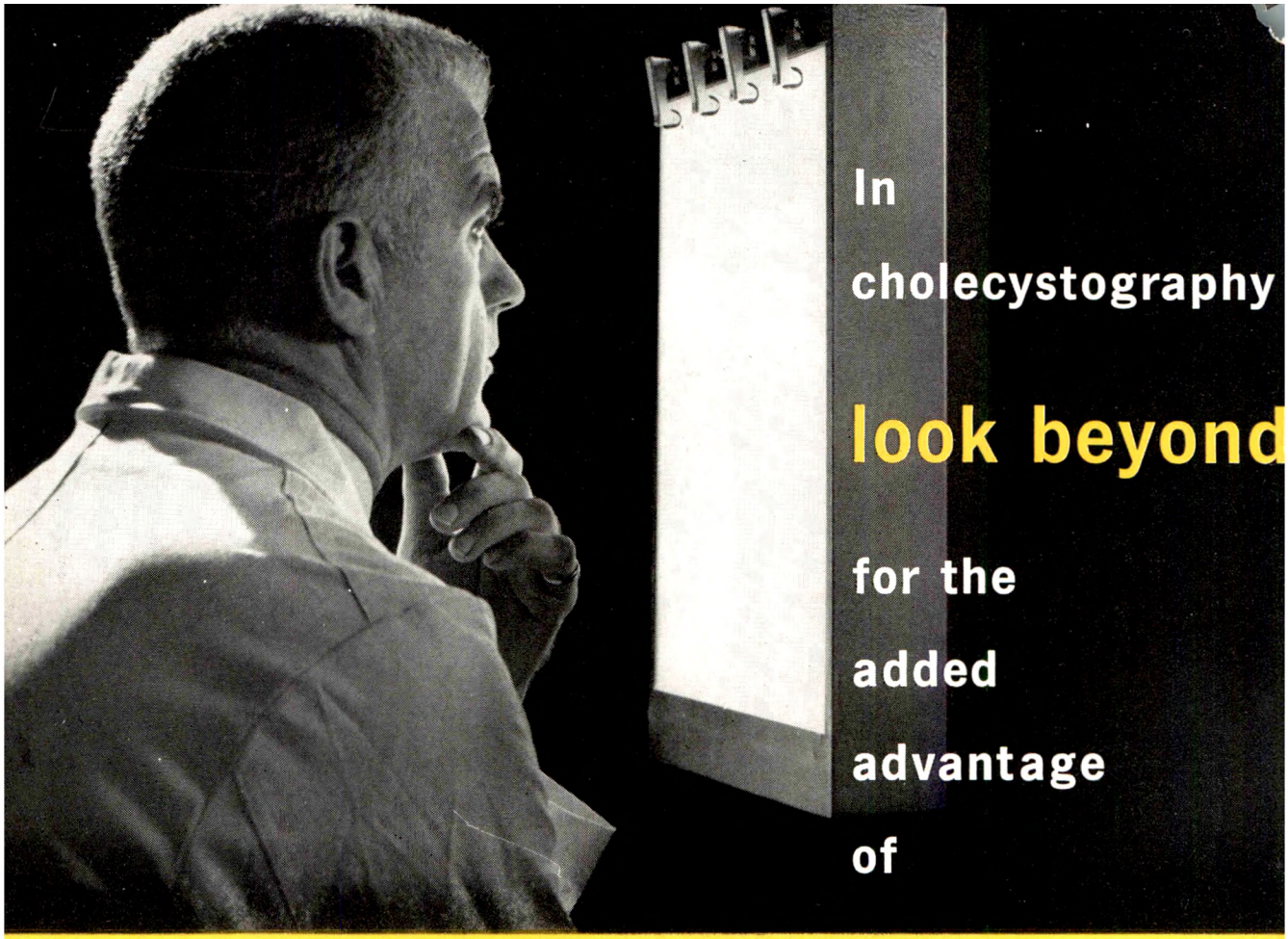
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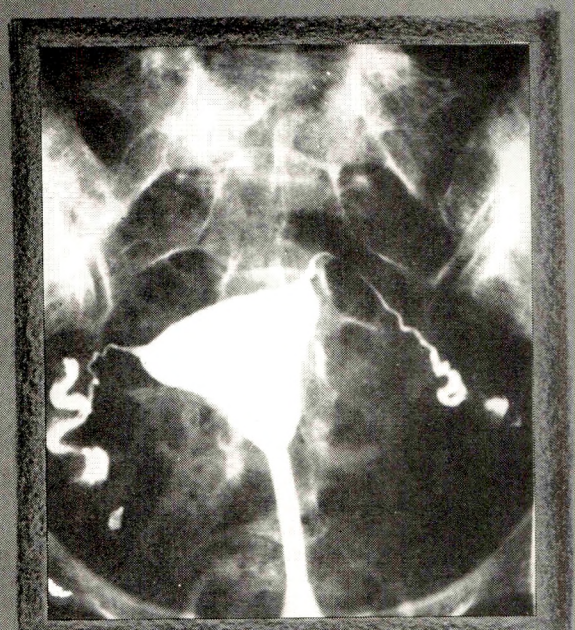
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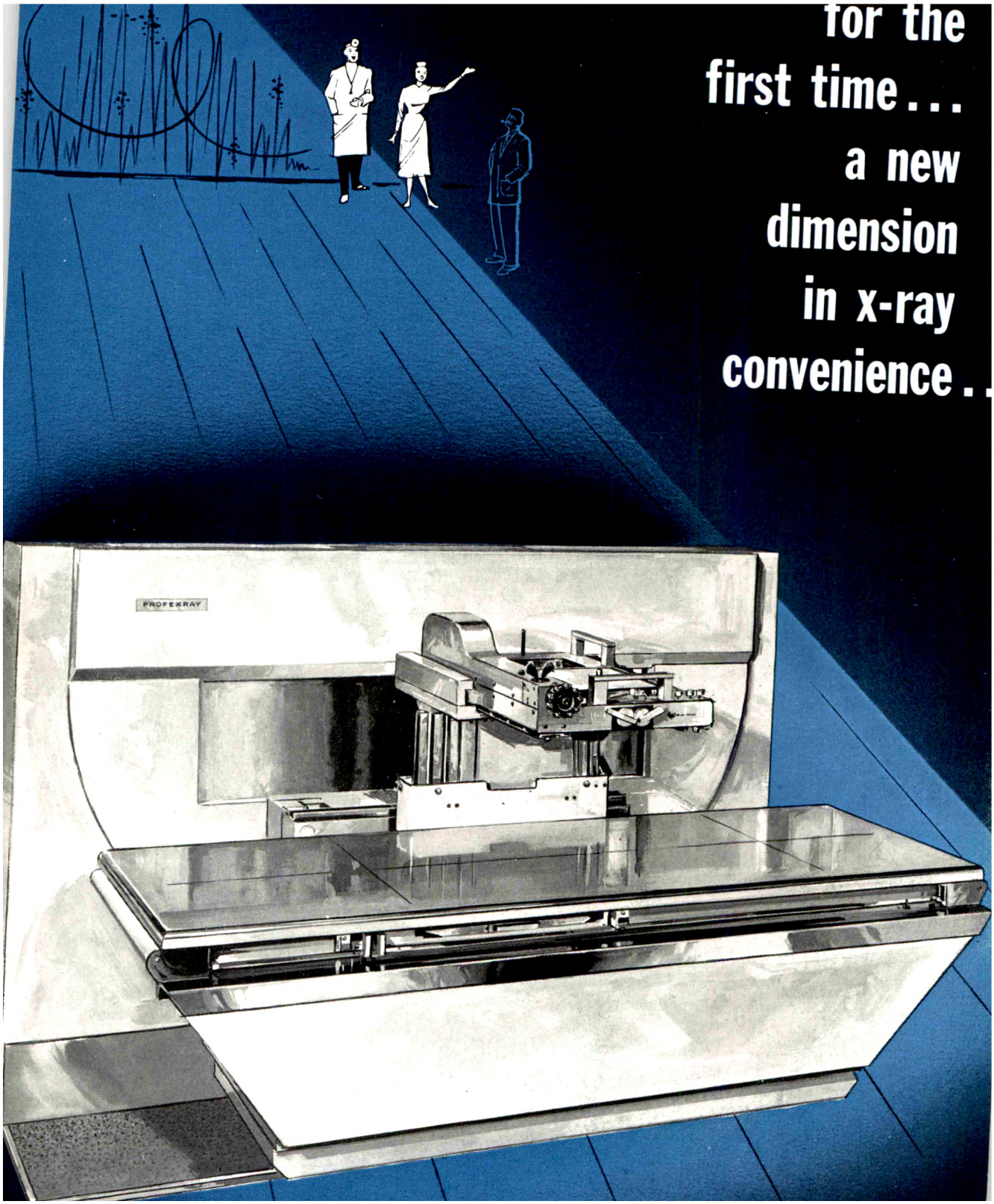

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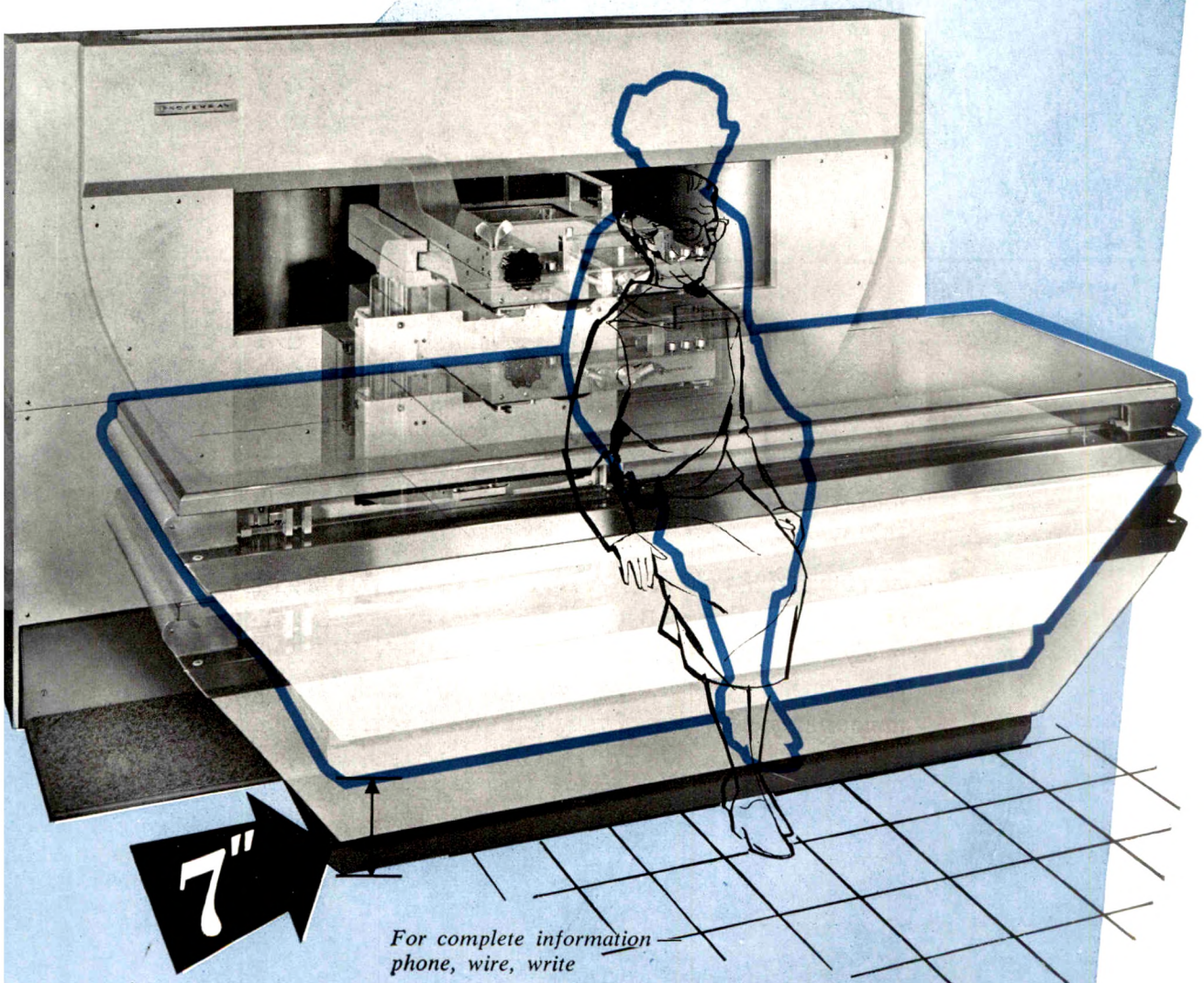
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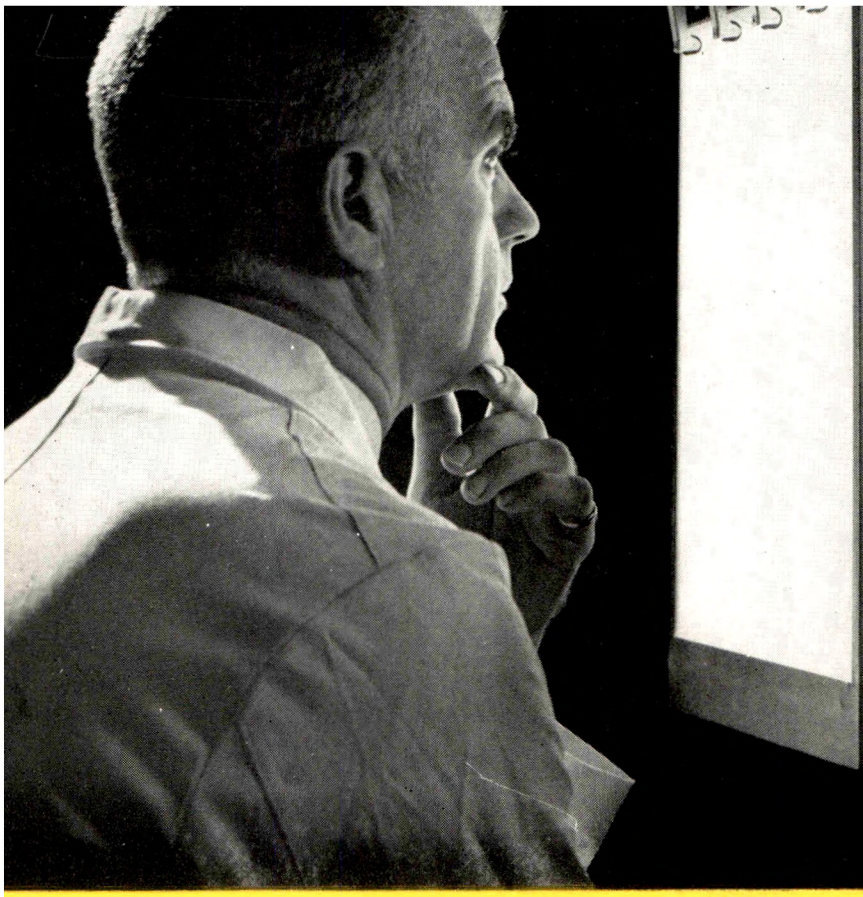
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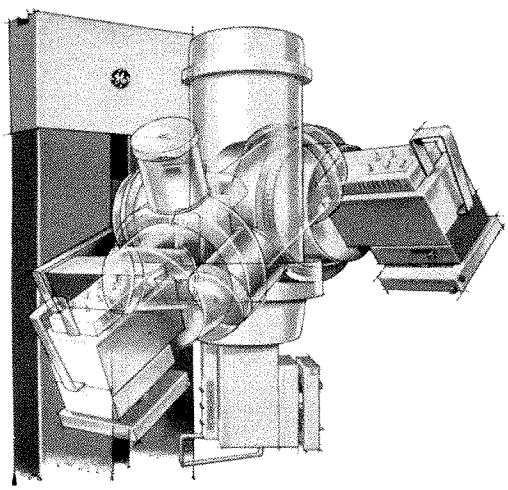
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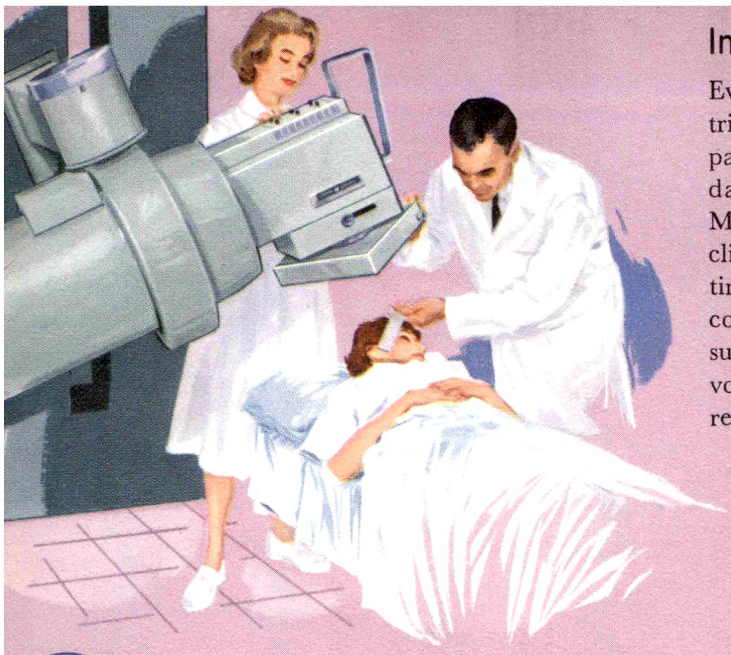
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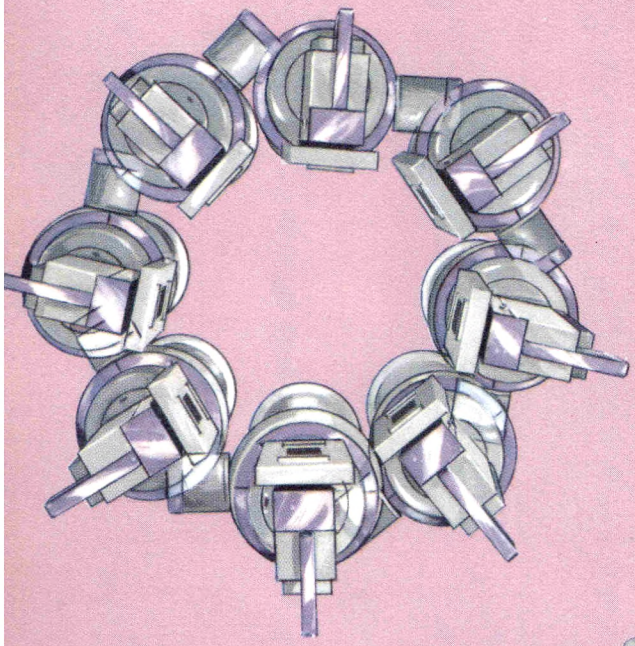


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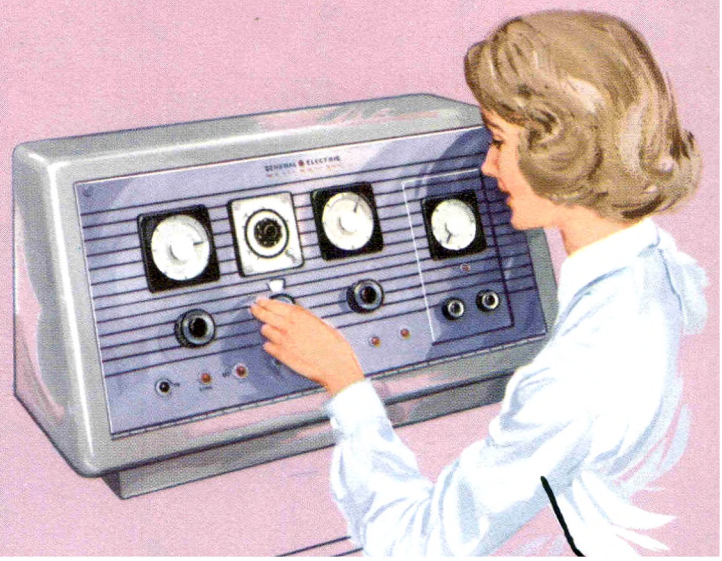


All-around operating flexibility

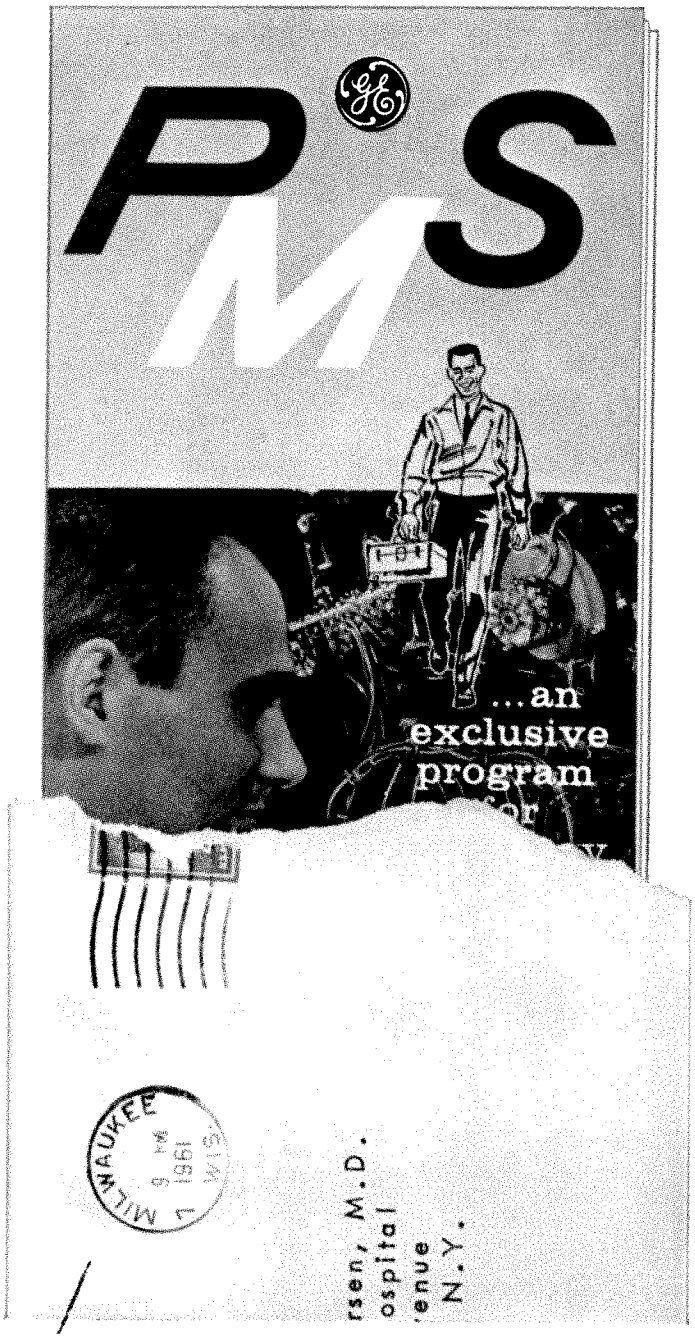
Whether you mean "handling ease" when you say flexibility—or are thinking of freedom to position without limitation—Maxitron 300 has it! Up, down, in, out, across and around . . . its compact, gas-insulated tubehead responds smoothly and easily to guidance from your hand . . . locks "on target" with the flip of a switch. Such ease of adjustment greatly facilitates its use for intracavity, multiport or rotational applications.

Remote technic selection

Dial the exact dose rate and half-value layer you want, any of *ten* directly from control console (half-value layers range from 0.25-mm Al to 4-mm Cu). Correct filter spins into position, kvp and ma are matched—all automatically. Vernier adjustment of kvp and ma, registered on direct-reading meters . . . built-in beam monitor to check dose rate . . . precision, integrating-type, electric treatment timer . . . many other conveniences you'll appreciate in this compact control.



SPECIAL NEWS FOR OWNERS OF GENERAL ELECTRIC X-RAY EQUIPMENT



NOW YOU CAN SAVE ON SERVICE WITH YEAR-ROUND PLANNED MAINTENANCE!

G-E x-ray equipment owners everywhere now can share in a unique new program called *Planned Maintenance Service.* It tackles x-ray service problems from two angles: First, through regularly scheduled "tune-ups" of equipment . . . second, by providing emergency service any time during business hours, free of further labor charges.

Curb repair costs. Pay only the monthly rate and, thereafter, you're free of service-labor charges no matter how serious the problem or how frequently you request help!

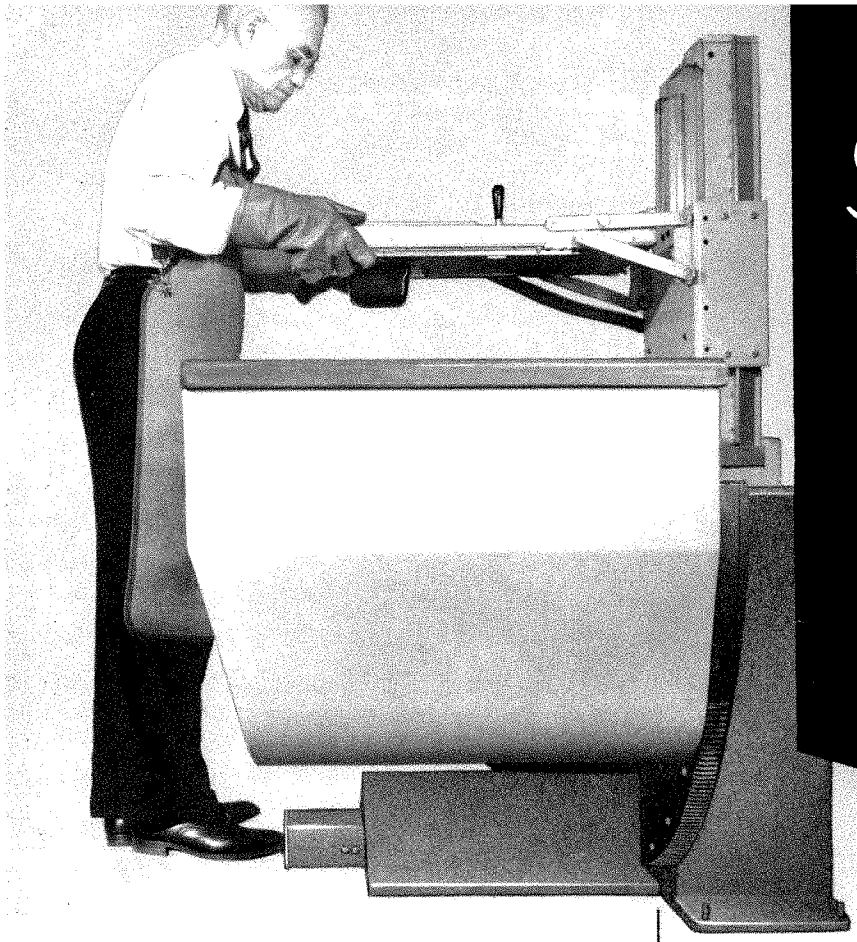
Extend equipment life. By detecting trouble early and correcting it, apparatus life is prolonged . . . premature deterioration forestalled. Performance stays at unusual levels of efficiency.

Easier budgeting. This bonus advantage stems from never being in the dark about future labor costs for x-ray repairs. Makes it easy to establish a figure for your annual budget.

Full details on *Planned Maintenance Service* can be obtained from your G-E x-ray representative. Or write to X-Ray Department, General Electric Company, Milwaukee 1, Wisconsin, requesting Pub. A-72.

Progress Is Our Most Important Product

GENERAL ELECTRIC



• *the ULTIMATE in table design!*

Standard

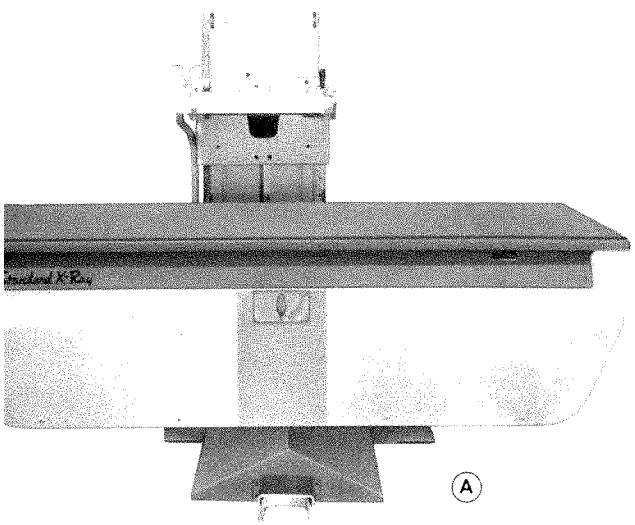
ULTIMA-105

HEAVY DUTY RADIOGRAPHIC-FLUOROSCOPIC

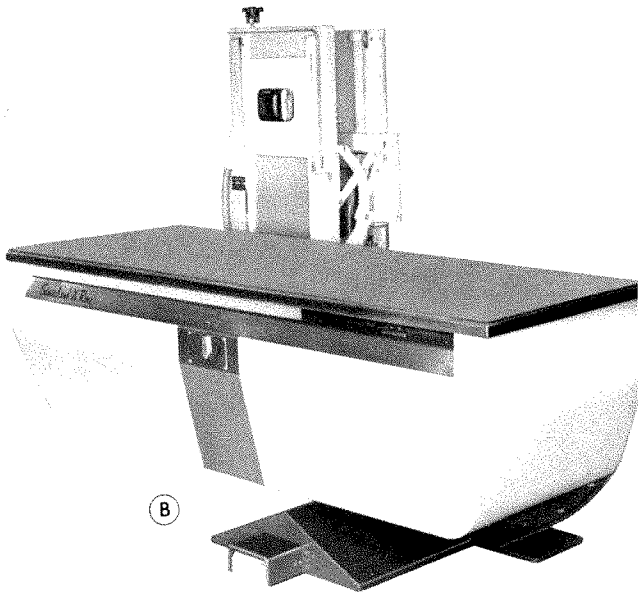
X-RAY TABLES

FOR THE PATIENT: *the ultimate in comfort*

FOR THE RADIOLOGIST: *the ultimate in efficiency and convenience . . . less work and less effort to do it!*



(A)



(B)

• WRITE FOR COMPLETE DETAILS

Ⓐ **FUNCTIONAL CONVENIENCE:** Maximum accessibility to the table. There's NO FRONT LEG, covered-up or exposed, that keeps the radiologist a full six inches away from the table and spot film device. He can now work comfortably right up against the table without back-breaking bending and strain, resulting in more ease and much less effort! Superior design makes it unnecessary for table top to move transversely.

FUNCTIONAL EFFICIENCY: The manually operated fluoroscopic collimator is very, very new! The proven best method of coning in radiography is now adapted for fluoroscopy as standard equipment, at no extra cost.

Ⓑ Spot film device is quickly, easily "parked" out of the way, leaving table top free and clear. Movement is smooth and light because of the linkage mount and weight-saving counterbalance method. Low profile of tower and spot film device in "parked" position eliminates interference with shock-proof radiographic cables.

Ⓒ Operates on a single fulcrum. Tilts 90° vertical, and not 10 or 12, but full 15° Trandelenberg. Automatic horizontal stop is standard equipment. Provides coast-free tilting from foot switch on base or hand switch on spot device.

Ⓒ Magnetic locks for all movements of the fluoroscopic and spot film staging. Switch control at operator's fingertips or automatic operation by the spot device action.

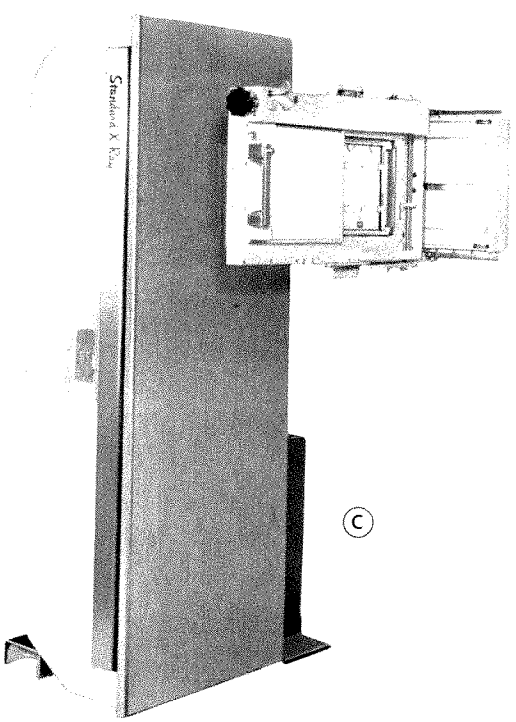
FUNCTIONAL BEAUTY: A truly handsome table with clean, simple lines. No unnecessary decoration to increase cost or decrease function. It's good looking in a dignified way. Smooth surfaces are easy to clean and keep clean.

Smart two-tone colors: light sand tone on the body to emphasize working area; dark hammer tone on base that continues to look good after years of cleaning, sweeping, spillage or just plain abuse.

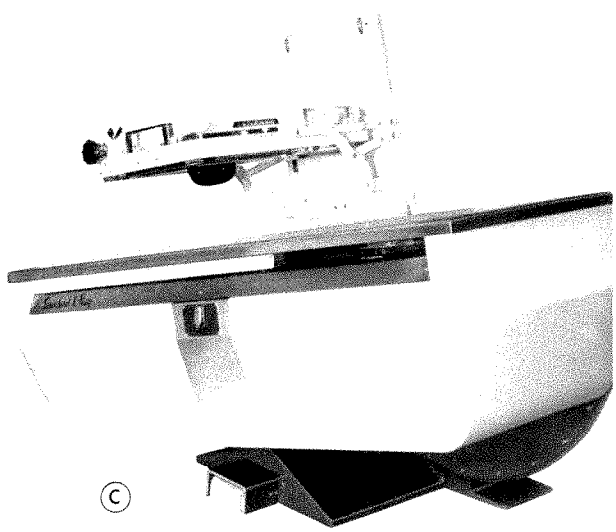
BUILT-IN PROTECTION: Adequate protection is a "must" and this table certainly has it! Completely enclosed body and automatic bucky slot cover give ample protection. A retractable radiation shield is optional equipment.

YEARS OF TROUBLE-FREE SERVICE: This is a first line, heavy duty table designed for the exacting radiologist, and built to stand up under the heaviest hospital service. Why? because it has the largest bearings of any table on the market, the best materials and the simplest mechanisms throughout. It is the finest example of Standard's 50 years of experience in the field.

TOP VALUE! This table is all new from the ground up! It is a heavy duty, quality table, that feature-for-feature provides more basic value for the price! Others may be lower priced (we make them, too), but none outshines this as a better buy!



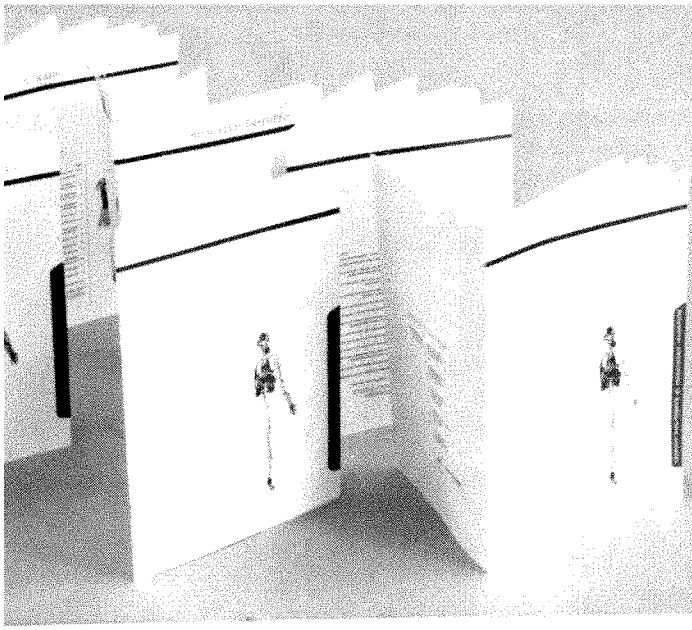
Ⓒ



Ⓒ

Standard

STANDARD R X Y



Revised edition now available from Nuclear-Chicago

DIAGNOSTIC APPLICATIONS OF RADIOACTIVE ISOTOPES

The newly published second edition of Diagnostic Applications of Radioactive Isotopes contains new radioisotope diagnostic procedures along with modifications of previously presented methods. The contents, listed to the right, suggest the broad scope of this useful and widely accepted publication.

Subject matter describes in outline form the most commonly used procedures for each application, and pertinent references will lead the reader to other methods.

Although the text is intended primarily for those unfamiliar with radioisotope applications to clinical medicine, many will find it a handy refresher guide in this area. Useful also in training programs, the publication provides a convenient reference to commonly used radioisotope diagnostic techniques.

Nuclear-Chicago will be happy to send you a copy without obligation.

nuclear-chicago
CORPORATION

Contents

How to establish your radioisotope program

Evaluation of thyroid function using radioiodine

Estimation of thyroid function with red cell uptake of radioiodine labelled thyronine

Determination of conversion ratio as an estimation of thyroid function

Evaluation of kidney function with radioiodine labelled compounds

Estimation of cardiac output with radioiodinated serum albumin

Estimation of myocardial blood flow

Blood and plasma volume measurements using radioiodinated serum albumin

Measurement of red cell mass with radiochromium

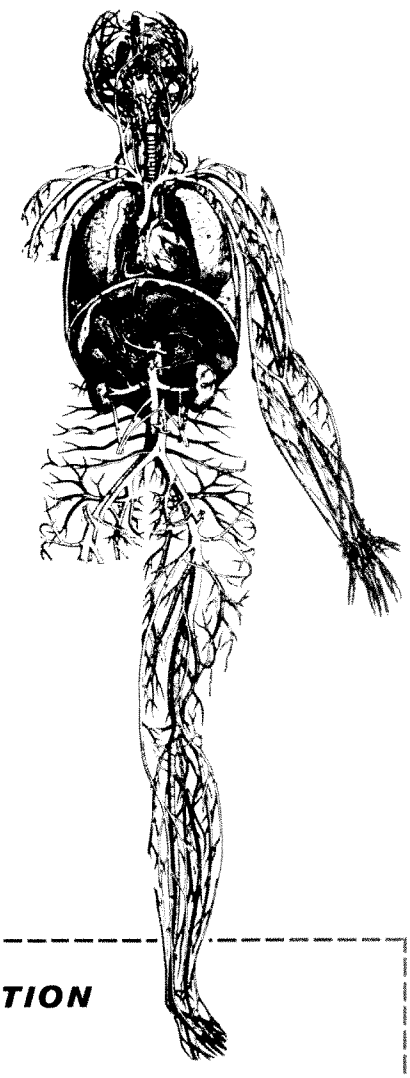
Measurement of red cell survival with radiochromium

Diagnosis of pernicious anemia with radiocobalt vitamin B_{12}

Measurement of fat digestion and absorption with radioiodine labelled triolein and oleic acid

Scanning body areas for radioisotope concentrations

Use of needle-type probe scintillation detectors



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OF RADIOACTIVE ISOTOPES

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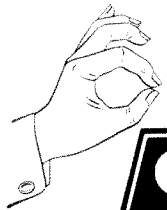
City Zone State



the Key

to reduced radiation exposure

Constant research, development and perfection in Gevaert laboratories today bring you as fine and fast a film as dedicated technicians can produce. Even with a sharp reduction in exposure, your Gevaert film will enlighten your view box with a radiograph of gratifying brilliance and clarity. Seeing is believing.

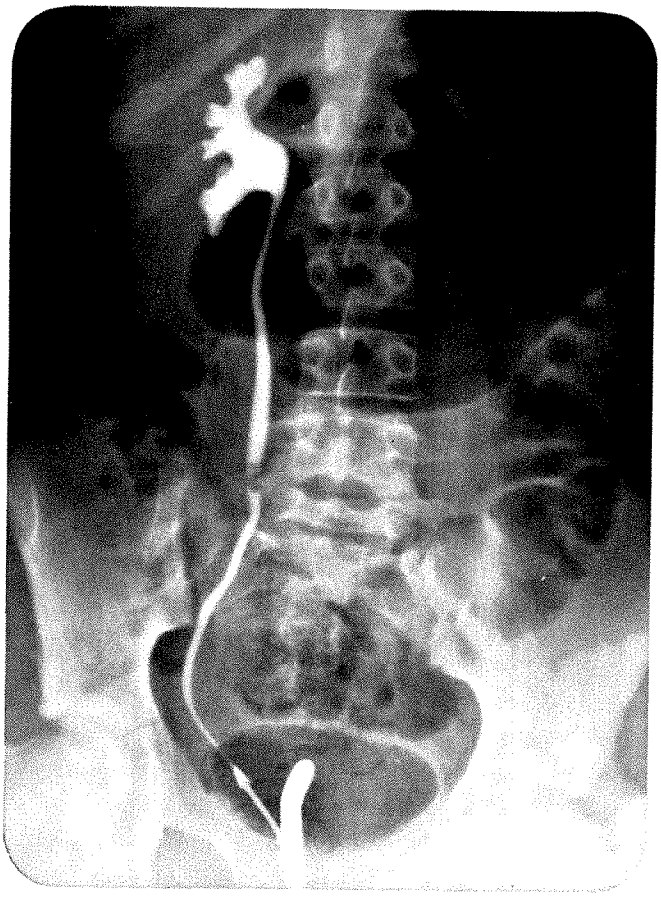


"NE PLUS ULTRA"

GEVAERT
X-RAY FILM

Exclusive Distributors: Low X-Ray Film Corporation, 161 Sixth Avenue, New York 13, N. Y. • Other Offices: 9109 Sovereign Row, Dallas 35, Texas • 725 No. Highland Ave., Los Angeles 38, Calif. • 6611 No. Lincoln Ave., Chicago 45, Ill. • Nationwide Warehousing

Retrograde pyelography "without serious reactions"[1]



Unlike other retrograde contrast media, which produce a high reaction rate (25-35%),[1] side effects are negligible with Retrografin—a unique agent that combines Renografin with neomycin, bactericidal agent of choice for addition to urographic media.[2] Efficacy and virtual absence of side effects have been amply confirmed in three reports covering a total of 340 patients.[1,3,4]

Clinicians are impressed by the diagnostic quality of films taken after Retrografin; well-defined radiographs, uniformly excellent in detail, have been consistently obtained.[1,3] Retrografin permits retrograde pyelography in patients with a known or suspected active urinary infection. Moreover, with Retrografin, new infection or spread of established infection does not occur.[1,3,4]

Retrografin

Squibb Diatrizoate Methylglucamine Injection U.S.P. with Neomycin Sulfate U.S.P.

Supply: Retrografin is supplied in 25 cc. and 50 cc. vials containing Renografin 30% with 2½% Neomycin as the sulfate. *Also available:* ***Renografin-30*** in vials of 50 cc.

For full information, see your Squibb Product Reference.

'RETROGRAFIN'® AND 'RENOGRAFIN'® ARE SQUIBB TRADEMARKS.

References: (1) Brimberg, J.; Berlowitz, H. D.; Schwinger, H. N.; Renner, M. J.; Lopyan, M.; and Beilouny, R.: New York State J. Med. *59*:2186 (June 1) 1959. (2) Roth, R. B.; Kaminsky, A. F.; and Hess, E.: J. Urol. *74*:563 (Oct.) 1955. (3) Bloom, J., and Richardson, J. F.: J. Urol. *81*:332 (Feb.) 1959. (4) Samellas, W.; Biel, L., Jr.; and Draper, J. W.: New York State J. Med. *59*:2570 (July 1) 1959.

SQUIBB *Squibb Quality—the Priceless Ingredient*

SQUIBB DIVISION Olin N.Y.

NEO-CHOLEX

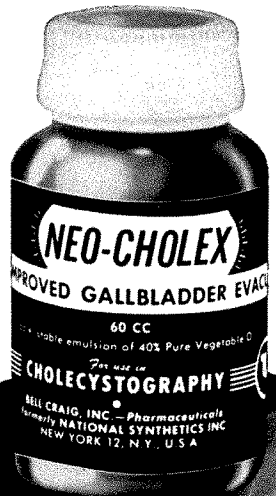
IN CHOLECYSTOGRAPHY

the Modern, Rapidly Acting, Palatable, Concentrated Fat Meal Always Ready at Hand for Immediate Convenient Administration

The fat meal in cholecystography is traditional for its efficacy in aiding disclosure of gallbladder anomalies, small stones, polyps, etc.

However, modern requirements call for detection, as well, of abnormalities of the biliary tree.

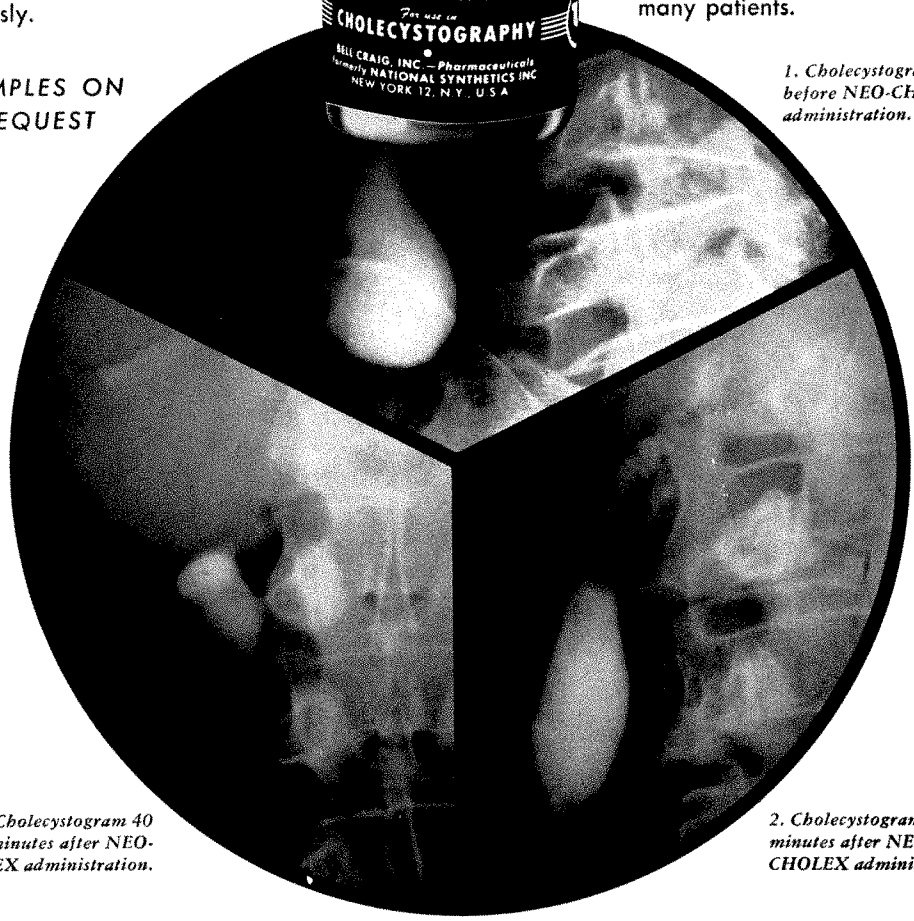
NEO-CHOLEX, the highly-concentrated vegetable fat emulsion accomplishes *both* results simultaneously.



Within minutes after ingestion, delineation of the ducts (cystic, common and at times hepatic) is successfully accomplished without the hazards of intravenous injections.

Neo-Cholex is palatable, requires no refrigeration. It also avoids the nuisance of a fatty breakfast with its attendant disadvantages of bloating, nausea and aversion to such foods by many patients.

SAMPLES ON REQUEST



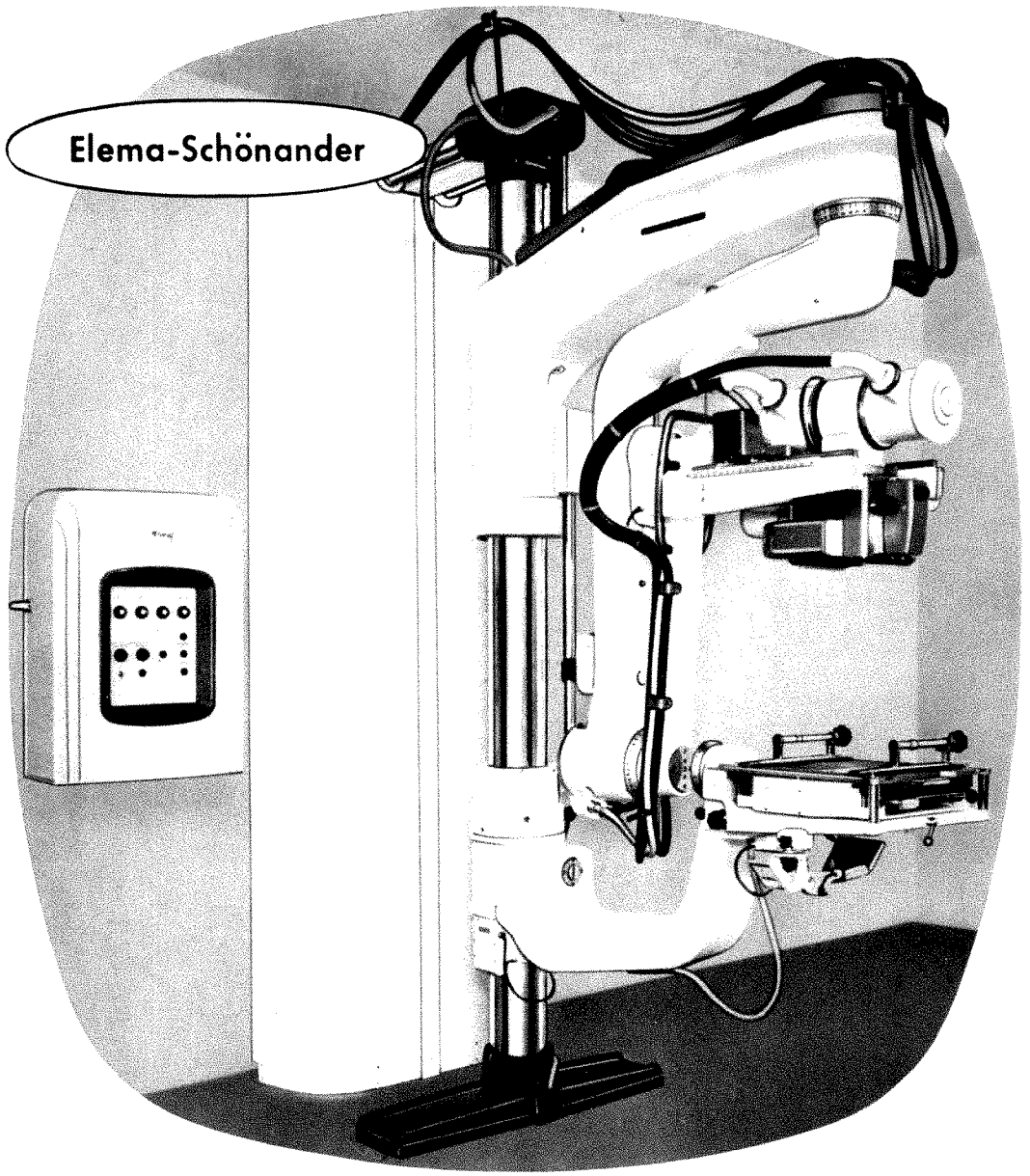
1. Cholecystogram before NEO-CHOLEX administration.

2. Cholecystogram 8 minutes after NEO-CHOLEX administration.

3. Cholecystogram 40 minutes after NEO-CHOLEX administration.

BELL-CRAIG, INC. 270 Lafayette Street, New York 12, New York

In Canada: GILBERT & CO., LTD., Toronto, 4



Mimer Universal X-Ray unit, designed for Neuroradiology and other special radiographic work.

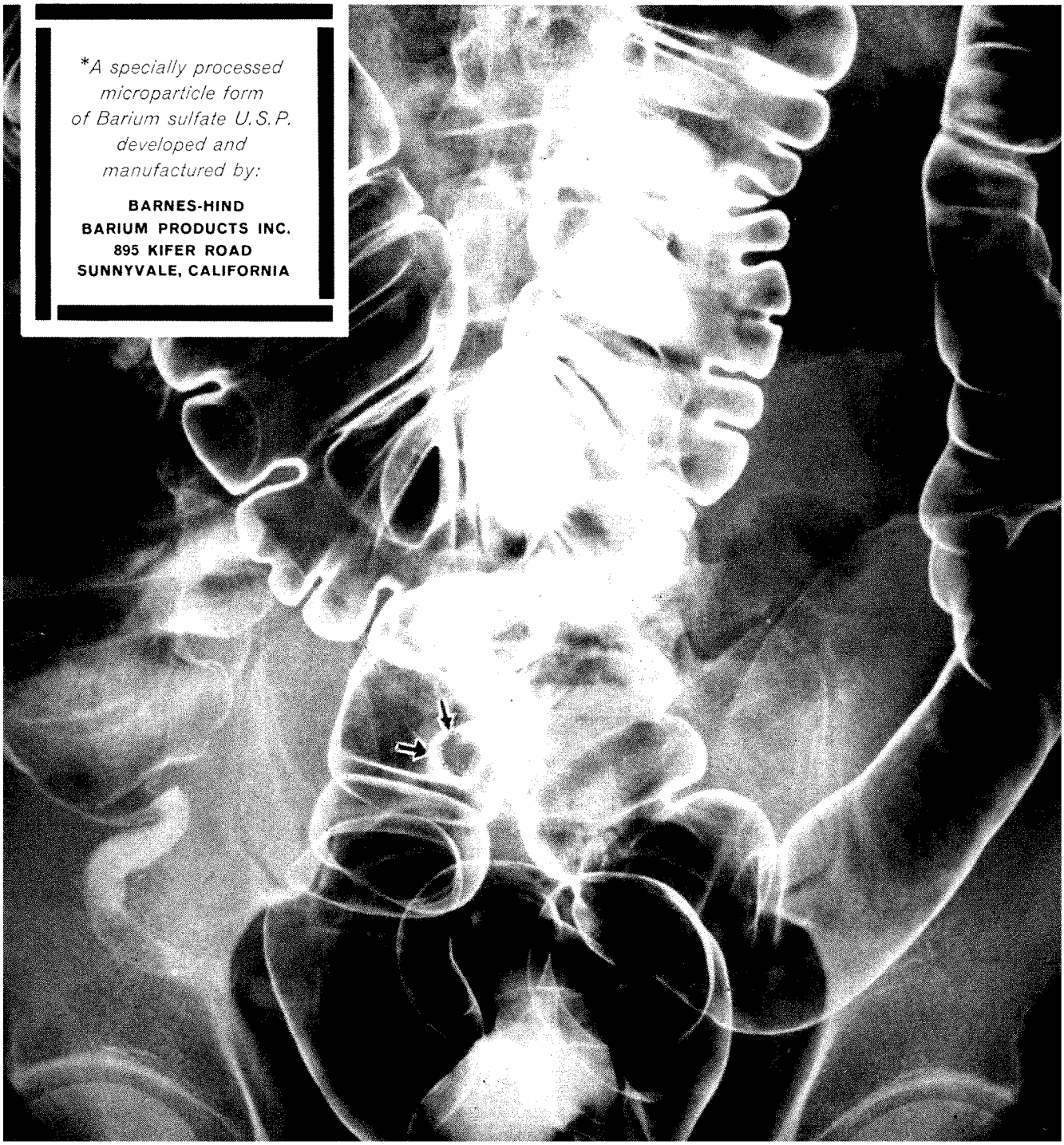
For details contact your local dealer.

SCHICK X-RAY company, inc.

444 NORTH LAKE SHORE DRIVE · CHICAGO 11, ILLINOIS

for double contrast studies—

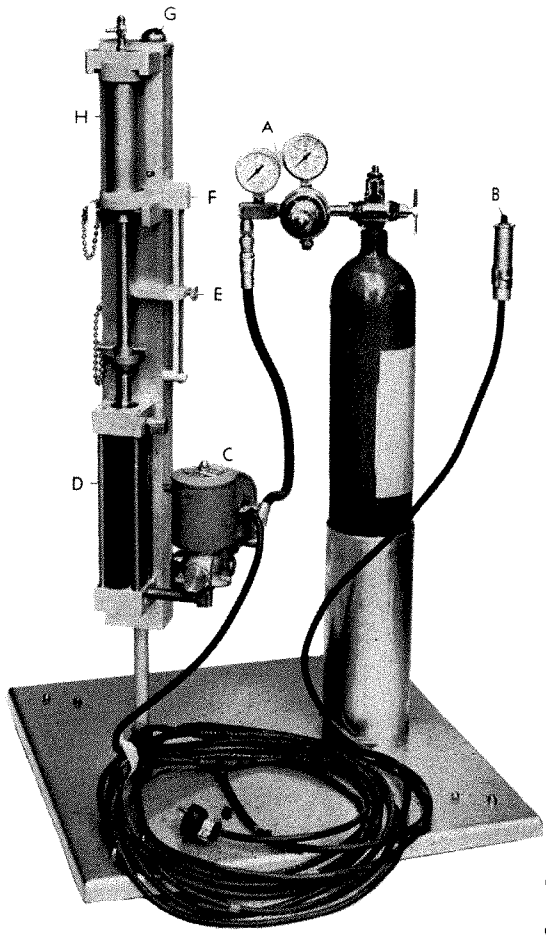
BAROTRAST** RESULTS LIKE THIS MAY BE OBTAINED ROUTINELY WITH **BAROTRAST. ■ THE UNPARALLELED FLOW, COATING, AND WATER RETENTION PROPERTIES OF BAROTRAST ARE NOT AFFECTED BY MUCIN, ENZYMES OR CHANGE IN pH. ■ BECAUSE BAROTRAST'S UNIFORM, STABLE, FLEXIBLE COATING WILL NOT CRACK OR PEEL, IT ADHERES UNINTERRUPTEDLY TO THE INTESTINAL MUCOSA. ■ AVAILABLE IN 25-LB. CARTONS FROM ALL RADIOLOGICAL SUPPLY HOUSES. COMPLETE PROFESSIONAL LITERATURE AVAILABLE.



**A specially processed microparticle form of Barium sulfate U.S.P. developed and manufactured by:*

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THE NEW PHELAN PHASIC DYE INJECTOR



is ready to help you by demonstrating its superior quality and simplicity of construction. Use this piece for thirty days and convince yourself that it is the best value on the market today in a phasic dye injector.

The component parts of the injector are:

- **A. Pressure regulator gauge.**
- **B. Momentary switch to energize solenoid valve.**
- **C. Solenoid switch operating on 115 V.**
- **D. Air cylinder—power source.**
- **E. Adjustment for graduating amount of media expressed.**
- **F. Micro switch to deenergize solonoid valve.**
- **G. Pilot light operating simultaneously with syringe.**
- **H. Syringe. 100 cc capacity furnished with unit. (50 cc capacity available at extra cost.)**

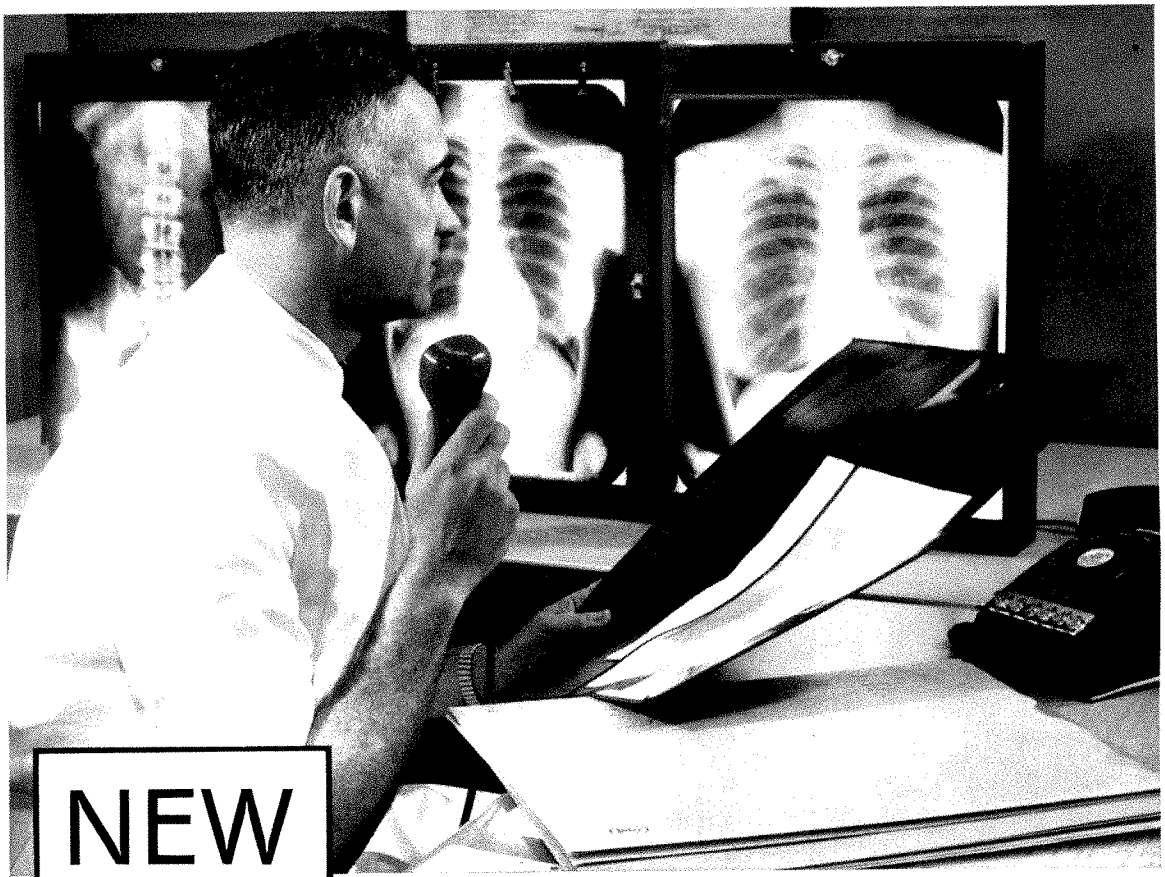
CO_2 gas recommended for use. ***Never use oxygen.***

The complete unit is adjustable for height on its vertical standard. Mounted on steel base with baked enamel finish—supported by four conductive castors.

Price—$440.00, f.o.b. Minneapolis. Includes everything pictured with the exception of the pressure bottle.

For further information write or phone collect

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whenever and wherever radiographs move

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protects them . . . keeps them together with patient records . . .

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Sturdy, stain-proof plastic construction with durable nylon stitched seams. Available in economical lots of 20 from your Ansco dealer.



For further information, write ANSCO, *A Division of General Aniline & Film Corp., Binghamton, N. Y.*

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SCALERS AT SENSIBLE PRICES

Among these new scalers, there's one (or more) precisely tailored to your need. Common to all are . . .

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precision pulse height analyzer or discriminator (*optional*)
automatic operation
count and time read-out (*optional*)
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(because they're *fully* transistorized)

greater operating stability
greater long term stability
no need for warm-up delay
longer component life
greater compactness
easier circuit replacement
more uniform production quality

prompt delivery

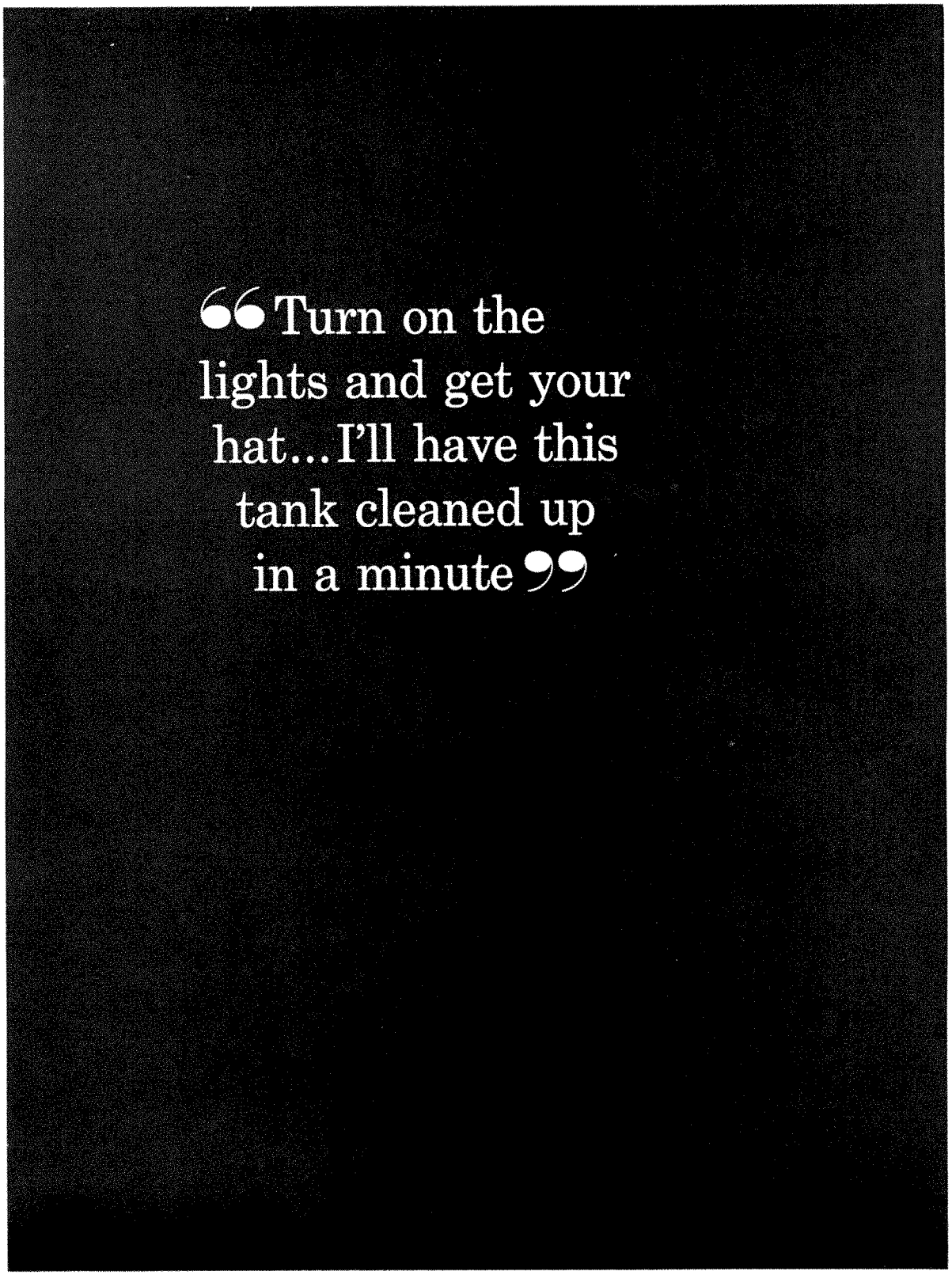
get details from your local Picker representative, or write Picker X-Ray Corporation, White Plains, New York



Picker Nuclear



no capital investment if you'd rather rent—ask about the PICKER RENTAL PLAN


“Turn on the
lights and get your
hat... I'll have this
tank cleaned up
in a minute”

Easier to clean... cost less...last longer!

G-E glass-fiber tanks for x-ray processing

JUST A MINUTE — that's all it takes to put, or keep, a G-E glass-fiber x-ray processing tank in tip-top condition. No need to spend time — and elbow grease — rubbing and polishing to remove dried chemicals or spots. The smooth, nonporous surface is immune to rust and corrosion. This tank won't warp or buckle from hot water. Basic cost is less than stainless steel . . . additional savings come from "packaged plumbing" which greatly reduces the cost of installation.

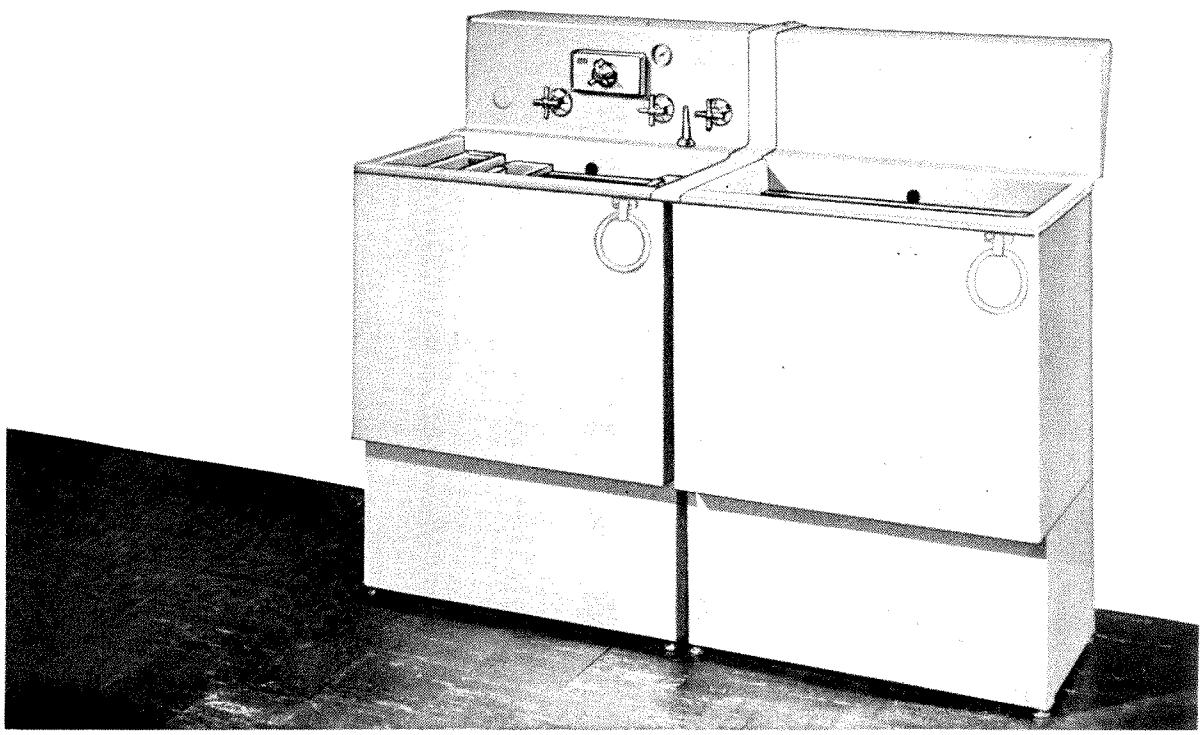
All of these built-in benefits are available from G.E. in a wide selection of tank sizes and types. Your choice of insulated and noninsulated models, 30″ and 48″ lengths, with or without pass hood, single compartment in either size, two- and three-compartment models in the 48″ size.

FULL LINE INCLUDES STAINLESS STEEL

To give you the widest possible choice General Electric master and pass tanks are also available in durable stainless steel. See your G-E representative for the details. Or write X-Ray Department, General Electric Company, Milwaukee 1, Wisconsin, for Pub. A-76.

Progress Is Our Most Important Product

GENERAL ELECTRIC

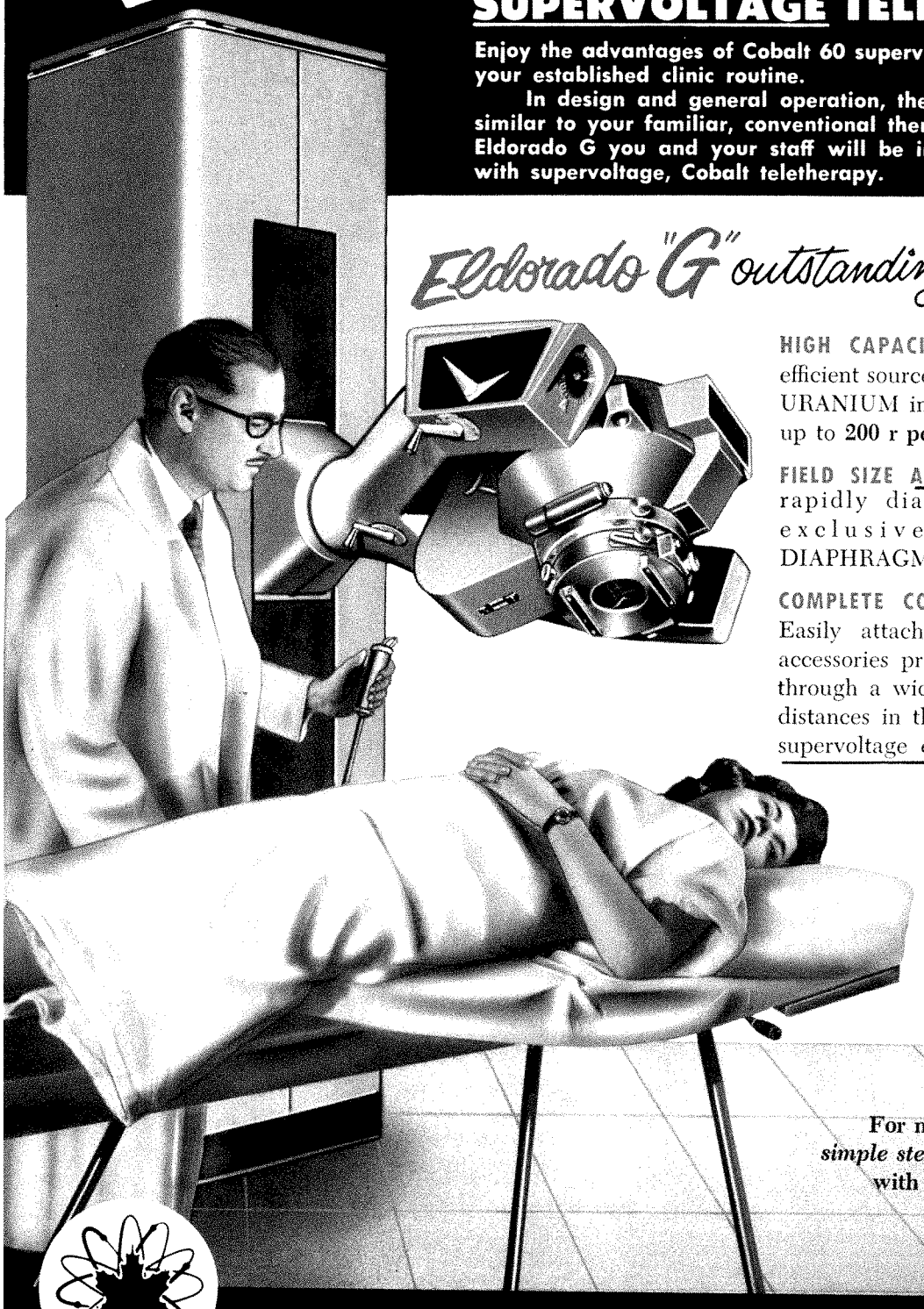


WITH THE Eldorado "G"

IT'S A SIMPLE STEP TO LOW COST SUPERVOLTAGE TELETHERAPY

Enjoy the advantages of Cobalt 60 supervoltages, yet maintain your established clinic routine.

In design and general operation, the Eldorado G is very similar to your familiar, conventional therapy units. Using the Eldorado G you and your staff will be immediately at home with supervoltage, Cobalt teletherapy.



Eldorado "G" outstanding features...

HIGH CAPACITY—with a compac efficient sourcehead design. An option URANIUM insert provides for outpu up to **200 r per minute at 50 cm. SS**

FIELD SIZE AND SHAPE SELECTION· rapidly dialled with A.E.C.L. exclusive, EIGHT SIDE DIAPHRAGM.

COMPLETE COLLIMATION CONTROL· Easily attached penumbra trimmi accessories provide well defined fiel through a wide selection of treatme distances in the medium and long, supervoltage equivalent ranges.

FULL POSITIONIN FLEXIBILITY—using du plane head swivel f full range efficiency set-up and treatment

For more details on the *simple step to supervoltages* with the Eldorado "G", please write to:

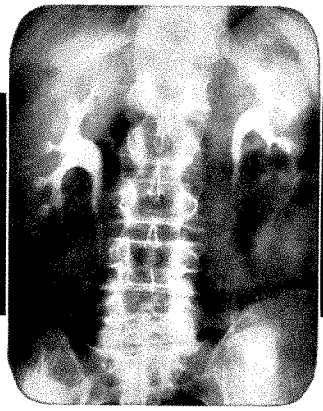
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HYPAQUE (BRAND OF DIATRIZOIC ACID), TRADEMARK REG. U.S. PAT. OFF.

New Instrument Measures Beta, Low-Energy X-Rays

CLEVELAND — A portable, vibrating reed, low-energy survey meter of extreme sensitivity for detection and measurement of beta and gamma radiation over a broad energy range was shown here today by the Victoreen Instrument Company.

According to a company spokesman, the new Victoreen Model 440 Low Energy Survey Meter brings greater safety to gamma ray surveys because it includes the often dangerous scatter components in the measurement.

Additional new applications pointed out for particular attention were X-ray crystal spectrometers, dental X-rays, radar sites, etc.

Victoreen, the pioneer designer and producer of radiation detection and measurement instruments, states the Model 440 has an accuracy of ±10% with gamma energy dependence of ±15% from 6.5 Kev to 1.2 Mev.

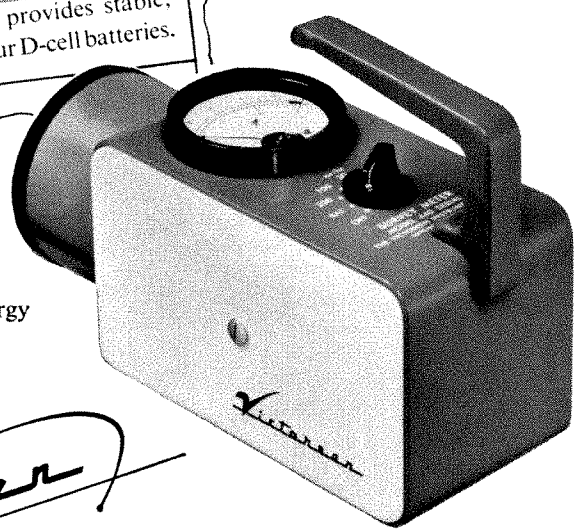
A single knob control turns the instrument On, provides five full-scale sensitivity ranges of 0-3, 0-10, 0-30, 0-100, 0-300 mr/hr. Transistorized and printed circuitry provides stable, trouble-free operation from four D-cell batteries.

Instrum...

AEC Sets New Lower Radioactivity Limits

WASHINGTON — The Atomic Energy Commission has sharply cut permissible radiation exposure standards, according to a statement issued here.

The new lower limits — the first change in the regulations in 3 years — will reduce lifetime accumulated dose to one-third of the previously permitted amount. Affected by the order are workers in nuclear installations, X-ray and radar



Victoreen Model 440 Low Energy Survey Meter

Victoreen

WORLD'S FIRST NUCLEAR COMPANY

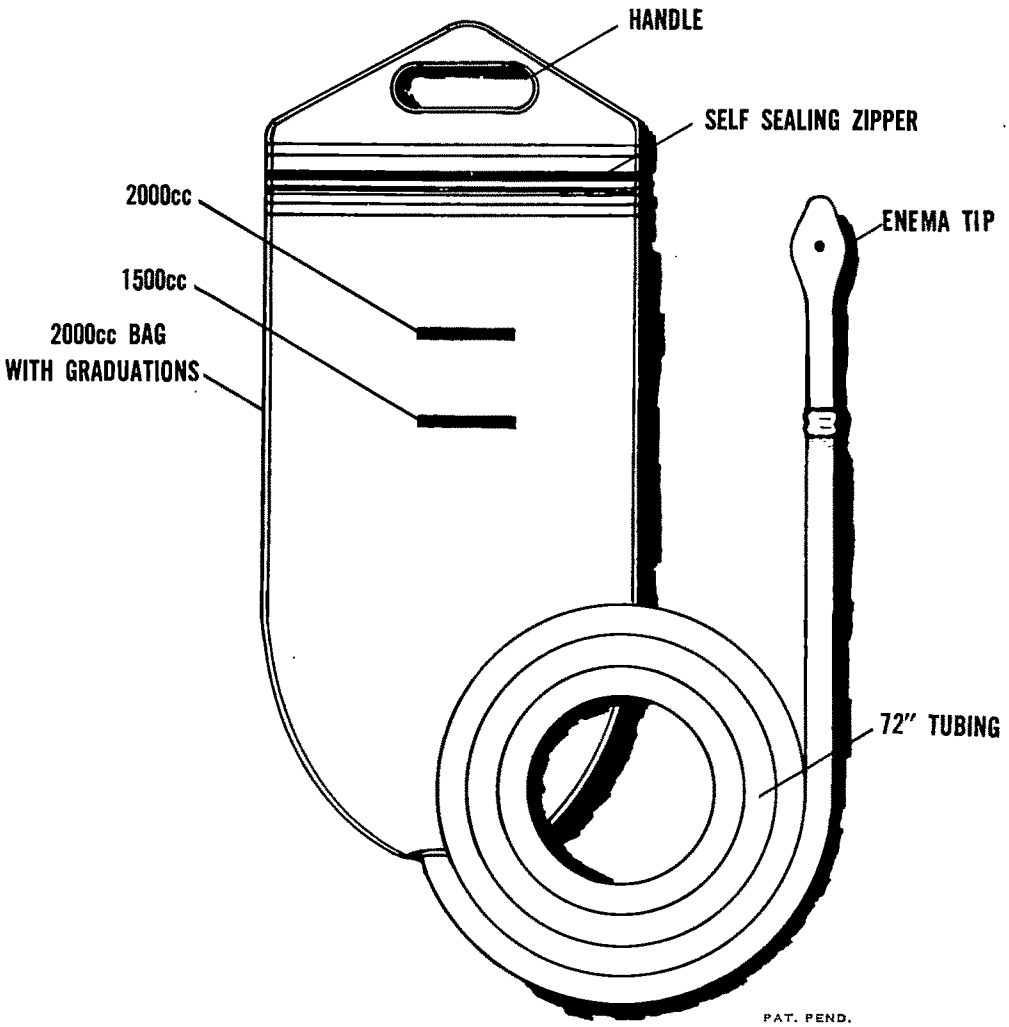
5806 HOUGH AVENUE • CLEVELAND 3, OHIO

A-4300A

The only one piece, inexpensive, completely disposable Barium Enema Bag in the market.

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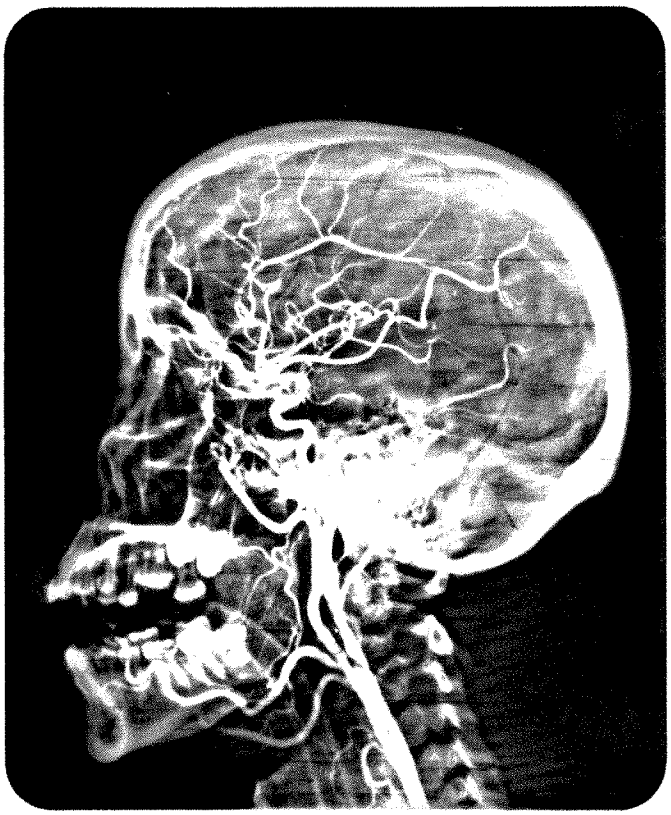
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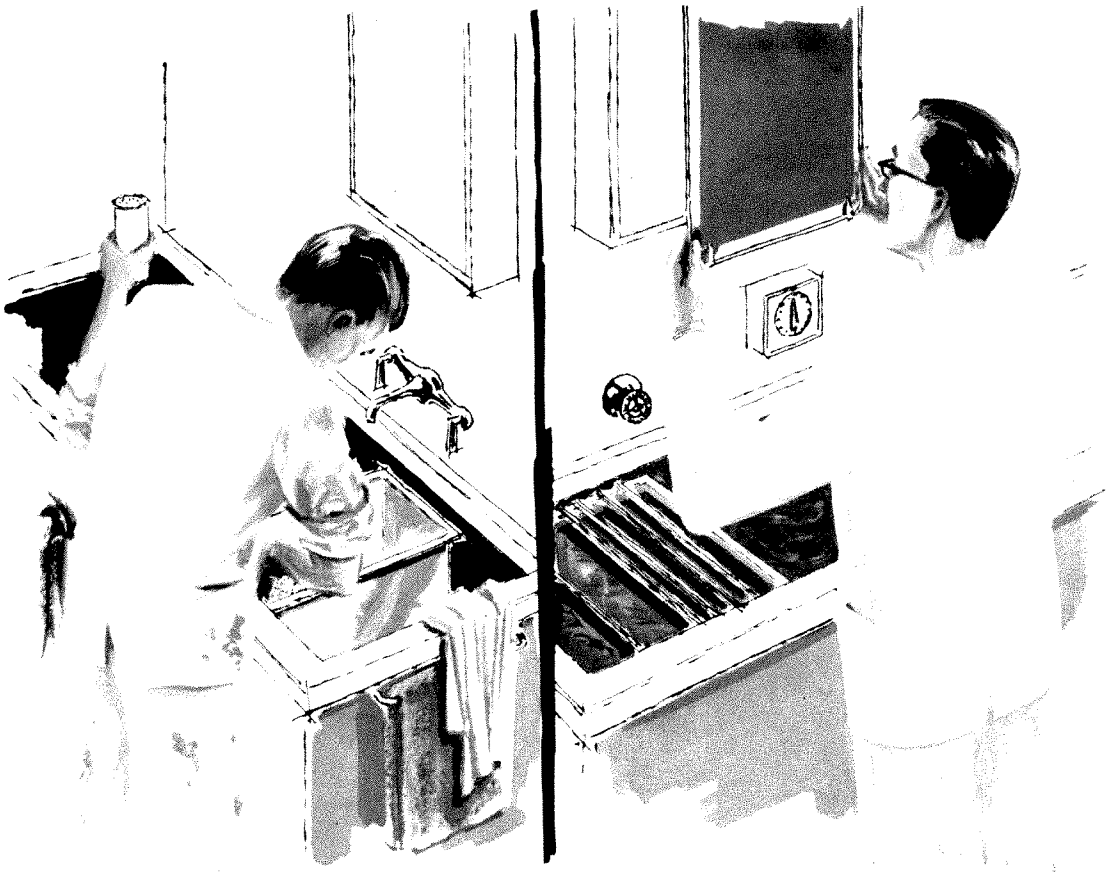
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References: (1) Doehner, G. A., and Brugger, G. E.: New York State J. Med. *60*:4022 (Dec. 15) 1960. (2) Gensini, G. G., and Ecker, A.: Radiology *75*:885 (Dec.) 1960. (3) Balkissoon, B.; Johnson, J. B.; Barber, J. B.; and Greene, C. S.: J.A.M.A. *169*:676 (Feb. 14) 1959. (4) Agnew, C. H., and Cooley, R. N.: Texas State J. Med. *55*:945 (Dec.) 1959. (5) Greenspan, R. H.; Bernstein, E. F.; and Loken, M. K.: Am. J. Roentgenol. *83*:1034 (June) 1960. (6) Kruse, C. A.: Western J. Surg. *67*:311 (Nov.-Dec.) 1959. (7) Barnett, J. C., and Arcomano, J. P.: Radiology *73*:245 (Aug.) 1959. (8) Utz, D. C., and Thompson, G. J.: Proc. Staff Meet. Mayo Clin. *33*:75 (Feb. 19) 1958. (9) Orr, L. M.; Campbell, J. L.; and Thomley, M. W.: J.A.M.A. *169*:1156 (March 14) 1959. (10) Mathews, P. W. Jr.: South. M. J. *52*:170 (Feb.) 1959.

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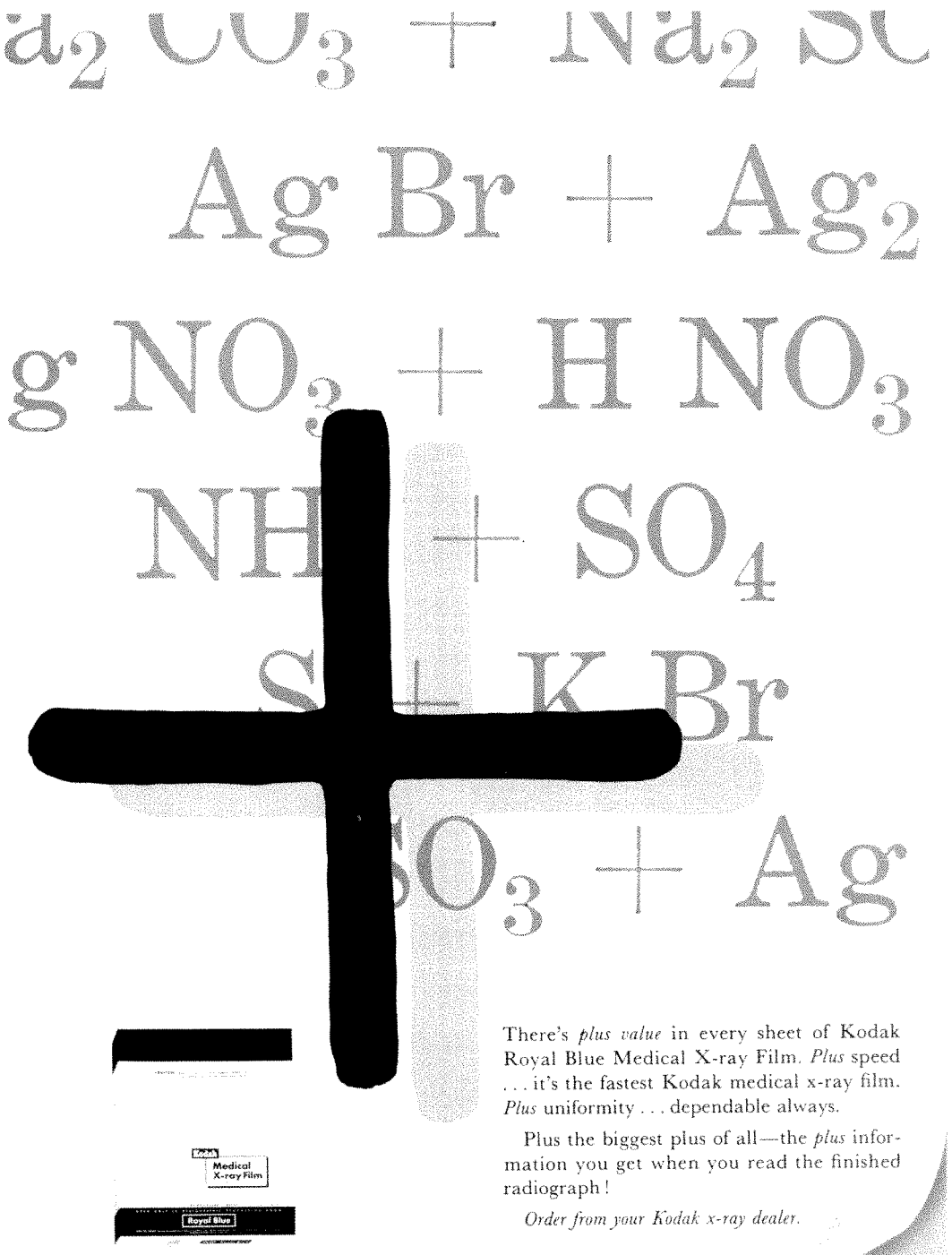
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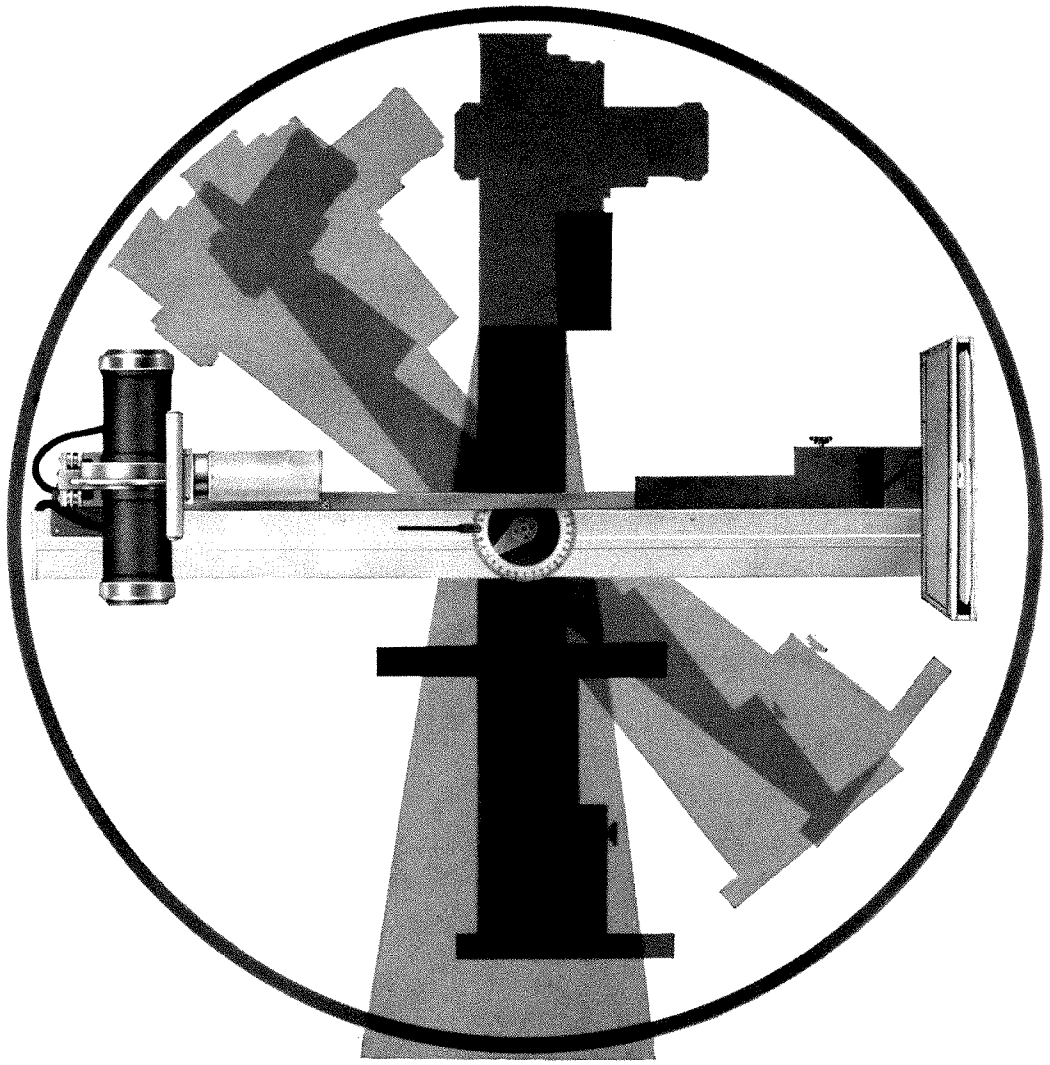
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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 86 JULY, 1961 No. 1

PANCREATIC CARCINOMA AND ITS EARLY ROENTGENOLOGIC RECOGNITION*

By JULIAN O. SALIK, M.D.†
BALTIMORE, MARYLAND

THE clinical diagnosis of malignant tumors of the pancreatico-ampullary area is often difficult and, because of their rapid growth, treatment in late stages quite unsatisfactory. Within the last several years a number of papers reported long term cures due to advances in operative techniques and the proved feasibility of radical pancreatico-duodenectomy. These long term results refer to neoplasms located in the head of the pancreas and adjacent duodeno-ampullary area since carcinomas of the body and/or tail either produce early metastases or are usually too advanced to be amenable to a successful operation. Therefore, an early recognition of pancreatico-ampullary neoplasms can prove to be life-prolonging, particularly so in the clinically nonjaundiced patient. These tumors can often be diagnosed by roentgenologic means on routine upper gastrointestinal studies when other clinical characteristics are still too indistinct, and the radiologist can thus play an important role in their early recognition. According to various reports, a correct preoperative roentgenologic diagnosis is made in about 30 per cent to 50 per cent of cases, including both the jaundiced and the nonjaundiced patients. In the jaundiced patient, there is—to a great extent—a "built-in" diagnosis, and usually in these cases the roentgenologic changes are obvious. With increased alertness and attention to the subtle, early roentgen signs, a correct preoperative diagnosis could perhaps be made in about 75 to 85 per cent of the cases, excluding tumors of the body and tail. In the latter location, it is difficult, if not impossible, by routine examination to detect the early roentgenologic signs of neoplasms. It should be emphasized that, although the roentgen manifestations might appear to be minor, the tumor may be found to be inoperable due to local extension or metastases. The location of the carcinoma is more important than its size, and small tumors arising near the duodenal wall may produce early roentgen changes as well as compression of the common duct with accompanying jaundice.

Excellent general reviews discussing the frequency, diagnostic criteria and the symptomatology of pancreatic neoplasms

* From the Departments of Radiology, Sinai Hospital and Johns Hopkins Hospital, Baltimore, Maryland.
† Radiologist-in-Chief, Sinai Hospital. Associate Professor of Radiology, Johns Hopkins University Medical School.

were published by Berk,[2] Cliffton,[7] Bank,[1] Johnson and Kalser[15] and Gullick.[13] The roentgenologic findings were described in great detail by Case,[5] Rendich *et al.*,[26] Kline and Culver,[16] Poppel,[24] Hodes *et al.*,[14] and others. In this paper, an attempt is made to emphasize the need for careful examination and familiarity with early, rather subtle, roentgenologic changes which could increase the preoperative diagnostic accuracy.

INCIDENCE AND LOCATION

According to statistics, carcinoma of the pancreas accounts for 1 to 4 per cent of all cancers, and about 17 per cent of cancers arising in the stomach, liver, biliary ducts and pancreas (Frantz[10]). It is said that carcinoma of the stomach occurs about four times as frequently as in the pancreas, but in our series this was not the case. During the years 1950 through 1959, 91 cases of carcinoma of the pancreas and 224 cases of carcinoma of the stomach were seen in our institution, corresponding to a ratio of 2.5:1. These neoplasms are more common in the male, the ratio being 2:1. They may occur in the younger age group, but the highest incidence is found between the ages of fifty to seventy. Carcinoma occurs in the head of the pancreas in about 65 to 80 per cent of cases; it is far less common in the body and tail, but sometimes it is difficult to establish the origin of the neoplasm as it might extend from the head into the body or involve the head and/or body and/or tail. Carcinoma is observed three times more frequently in the pancreas than in the papilla of Vater or the common duct. Tumors of the pancreaticoampullary area can arise from any of the several structures which are located in close proximity: the head of the pancreas, the common bile duct, the ampulla and papilla of Vater, the duct of Wirsung and/or Santorini, or the duodenal mucosa. A neoplasm arising in this area may envelop the surrounding structures to the extent that its exact origin is often obscured. As the long term cure rate of carcinoma of the ampulla of Vater and duodenum is much more satisfactory than that for carcinoma of the head, it is important that a preoperative or operative distinction be made.

LABORATORY DATA

According to Cattell *et al.*,[6] the majority of patients have elevated bilirubin values, ranging from 1.3 to 38 mg. per 100 cc.; in 50 per cent of their cases the serum bilirubin was below 10 mg. per 100 cc. and in 85 per cent below 20 mg. They stated that one should be hesitant with the diagnosis of carcinoma when the bilirubin value is less than 3 mg. or greater than 35 mg. We found that in the nonjaundiced patients the values were lower than 3 mg., but that, roentgenologically, changes were discernible which were compatible with the presence of a malignancy in the pancreatico-ampullary area. Alkaline phosphatase and cholesterol values are elevated, depending on the type of obstructive jaundice. In the beginning the values in flocculation tests are insignificant, but with recurrent cholangitis changes in flocculation values occur. Serum and urinary lipase and amylase may be elevated in patients when the pancreatic duct is blocked, but the values may be low if the obstruction has been prolonged with subsequent destruction of the secreting pancreas. Also, an investigation of the duodenal contents for pancreatic enzymes, sodium bicarbonate, occult blood and for cytologic changes may be of value.

PATHOLOGY AND SURGERY

A malignant tumor in the pancreaticoampullary area is usually a carcinoma and is rarely due to metastases, sarcoma, plasmocytoma, lymphosarcoma or Hodgkin's disease. Also, a malignant islet cell neoplasm (nesidioblastoma) or cystadenocarcinoma may be found. Benign tumors are usually adenomas or cystadenomas, dermoid cysts, composite tumors, nonepithelial benign tumors, or benign islet cell tumors (Frantz[10]).

Fraser[11] reviewed 1,035 cases of obstruc-

tive jaundice caused by pancreatic lesions and found that 74 per cent were due to neoplasms; 7 per cent were diagnosed as malignant although only an inflammatory disease was present, and in 16 per cent of cases pancreatitis was diagnosed when a neoplasm was present. Bowden[4] has pointed out that at times, at the operating table, it is often difficult to differentiate cancer from chronic pancreatitis, and that one may encounter a disparity between the size of the gross tumefaction in the head and that of the neoplasm which may be very small but seems larger because it is surrounded by a large inflammatory component. Biopsy is often avoided because of danger of pancreatitis, leakage, abscesses with fistula formation or peritonitis. Waugh and Gilbertson[29] recorded the results of operations on 85 patients up to January 1, 1953. Forty-nine traced patients survived three years or more, the rate being 28.6 per cent. Thirty-seven survived five years or more, the rate being 27 per cent. Nineteen patients were operated upon for carcinoma of the head and 3 lived five years or more, the survival rate being 15.8 per cent. Thirteen patients were operated upon for carcinoma of the ampulla and 5 lived five years, a rate of 38.4 per cent. Dennis and Varco[8] reported 2 patients with carcinoma of the head who survived six and nine years, and 3 with carcinoma of the ampulla who survived twelve, ten and nine years. Porter[25] reported 120 cases operated on between 1948 and 1952 and 130 between 1953 and 1957. In the first group 6 survived five years and 10 in the second. His patient's in general had carcinoma of the ampulla or duodenum. Millbourn[20] in Sweden reported his experience with 15 patients and found that the average survival rate in carcinoma of the head of the pancreas was twenty-three months as of September, 1958. Cattell *et al.*[6] recorded 148 patients who survived pancreatico-duodenal resection prior to February, 1958, and found that 1 out of 13 patients operated upon for carcinoma of the common bile duct was well. Fifty-five patients were operated upon for carcinoma of the ampulla of Vater, and of these 12 (36.8 per cent) lived five or more years; 13 patients were operated upon for carcinoma of the duodenum with 3 (37.5 per cent) surviving five years. Sixty-three patients were operated on for carcinoma of the head of the pancreas and of these 4 (9 per cent) survived for five years. Zintel *et al.*[31] reported 83 patients operated on for carcinoma of the pancreatico-ampullary area; 6 of these survived five to eleven years. None of the survivors was of the group in which the process began as carcinoma of the pancreatic acini.

SYMPTOMATOLOGY

The clinical signs and symptoms can be divided into early and immediate preoperative.

I. *Early Symptoms:*

A. Contrary to the widely held belief that painless jaundice is an early characteristic symptom, one of the earliest and most common complaints is pain which often precedes jaundice, and, in involvement of the body and tail, it may constitute the only symptom. The pain is usually not diagnostic, but three main types may be encountered: (a) a persistent, dull mid-epigastric pain radiating to the lower back, (b) pain, colicky in nature, usually in the right upper quadrant, extending to the subscapular area, and (c) a paroxysmal pain in the mid-abdomen radiating to the anterior part of the chest and back. Sometimes these patients visit doctor after doctor, and are labelled as psychoneurotics. Occasionally, they develop emotional disturbances, characterized by depression and anxiety, which can probably be traced to a fruitless search for medical help. The nature of the pain, its extent and location depend on the site of the tumor and the presence of local or distant metastases. In a series of 88 patients without jaundice, it was found that when the neoplasm involved the body and/or tail, the pain was generally on the left and in the back, while in tumors of the head the pain was usually on the right. In

carcinoma of the head, the pain is influenced by the activity of the upper gastrointestinal tract and is aggravated by intake of food, with relief following passage of stool or flatus. In involvement of the body and/or tail and, to a lesser extent, the head, relief from the pain can be obtained by a change in posture, as the pain seems to be more intense in horizontal position than in the upright or decubitus positions.

B. Another consistent symptom is weight loss. In carcinoma of the head, weight loss is more marked than when the papilla of Vater is initially involved, for in the latter jaundice occurs at an earlier stage. Weakness, anorexia, nausea, flatulence and vomiting are found in these patients.

C. Patients may complain of increasing constipation (which, in the older age group, is difficult to evaluate) and also of sudden onset of unexplained diarrhea with bulky stools primarily due to undigested fat and protein. This unexplained steatorrhea can be found in about 22.2 per cent of cases.

D. Gastrointestinal bleeding is noted in 25 to 37 per cent of cases, mainly in lesions of the duodenum and of the ampulla of Vater. However, it can occur in carcinomas of the head when, apparently, the tumor erodes and bleeds into the duct of Wirsung and thence into the duodenum.

E. Migrating thrombophlebitis, in our experience, was not a common occurrence, although the literature is replete with reports in this respect.

F. Pancreatic carcinomas occur more often in diabetics, but an abnormal glucose tolerance curve is frequently found in the nondiabetic patient.

II. *Immediate Preoperative Signs and Symptoms:*

A. Jaundice, usually progressive. Berk,[2] in his series of 1,361 cases, found that jaundice occurred in 64.7 per cent at some time. In carcinomas arising in the head, 81.3 per cent had jaundice, and in papillary neoplasms jaundice was present in all of the patients. Sometimes jaundice regresses temporarily due to necrosis and sloughing of a polypoid tumor about the papilla of Vater. Generally, in carcinomas of the head pain precedes jaundice; in ampullary malignancy jaundice might precede pain. Painless jaundice is relatively rare, and has been reported to occur in 8.8 to 26.5 per cent of cases in different series.

B. Pain, similar to that described in the early stage, but more relentless and severe.

C. Rapid weight loss with anorexia, marked weakness and pronounced gastrointestinal disturbances.

D. A palpable gallbladder is present in about 27 per cent of cases; enlargement of the common duct is more frequently present than the Courvoisier gallbladder (this applies more aptly to the common duct).

E. Enlarged liver (Schiff[27]).

F. Chills and fever.

ROENTGEN EXAMINATION

Over the past decade we have been interested in the possibility of improving the accuracy in diagnosis of pancreatic carcinomas by routine upper gastrointestinal examinations. Between January, 1950, and December, 1959, 91 cases of pancreatic carcinoma were operated upon or autopsied in our institution. Sixty-four of these were examined in our department and 49 recognized preoperatively; 47 were in the head, 10 in the head and body, 4 in the body, 2 in the tail and 1 in the entire pancreas with a large tumefaction of the tail. The last 6 cases were not clinically jaundiced. Eighteen of the carcinoma of the head cases were also not clinically jaundiced when examined, and 6 of these were reported as roentgenologically negative. Therefore, in our entire series we have had a 77 per cent diagnostic accuracy, and in the nonjaundiced neoplasms of the head a 68 per cent accuracy. The danger of false positive cases seems to be minimal, and we are still more concerned with under-reading rather than with over-reading. In our series, only 1 case was diagnosed as a questionable small tumor in the head of the pancreas producing a pressure below the

upper duodenal knee, and at surgery no evidence of any pathology was noted. In 2 instances, the tumor was found to represent pancreatitis and not a malignancy.

Because of its anatomic location (Fig. 1), a careful conventional upper gastrointestinal series may disclose pathologic changes in the head. In our experience, spot roentgenograms of the duodenal sweep in the erect position are not too valuable because the filling of the loop is inconstant and often not adequate for diagnostic purposes. The most useful views are serialographs—with or without the use of a pressure balloon—of the duodenum completely filled with barium and taken in the prone right anterior oblique position and prone horizontal position. Sometimes, the supine left posterior oblique position proves more useful (Wise and Johnston[30]). This is due to the fact that in the latter view the duodenal loop rests on the pancreas, and nodularities of the head may produce an imprint

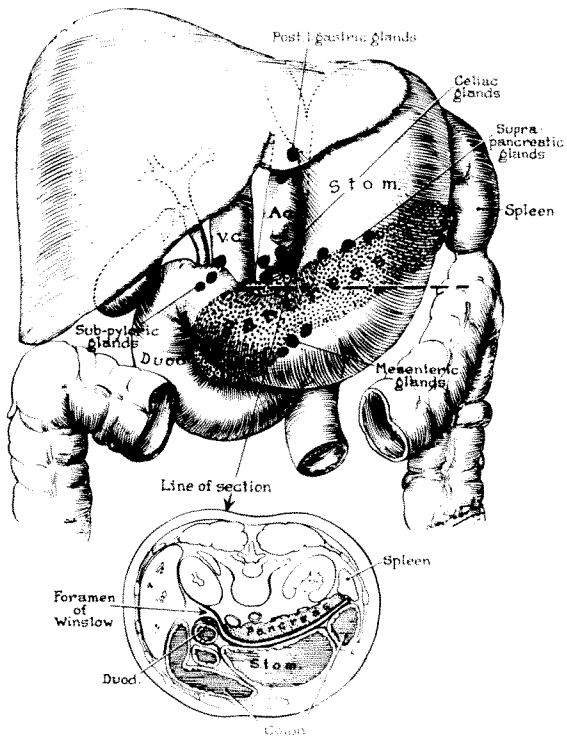


Fig. 1. Drawing showing the pancreas and its relation to surrounding structures.



Fig. 2. Roentgenograms showing normal duodenal loop with fine feathery or slightly coarser mucosal folds.

on the duodenum, whereas in the prone right anterior oblique position the duodenum tends to fall away from the pancreas. However, it may be difficult to obtain a satisfactory filling of the duodenal loop in the supine left posterior oblique position, and the prone views are usually quite adequate.

The following early roentgenologic findings are of significance:

1. Irregular contractions, occasionally marked spasticity of some segments of the duodenum. During fluoroscopy and on roentgenograms, a retroperistalsis and disordered motor function, especially of the mesial aspect of the second or third parts of the duodenum, are noted. The normal filling of the valvulae conniventes (Fig. 2) is present on the lateral aspect of the loop but altered or absent in some mesial segments of the duodenal sweep.

2. The mucosal folds in these segments of the inner aspect of the duodenal loop dis-

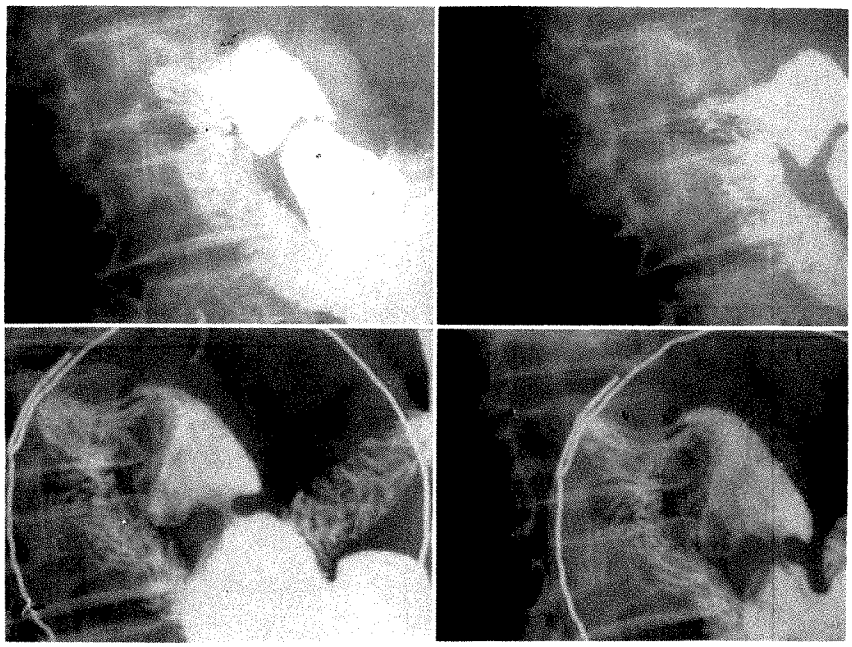


FIG. 3. B.G., a sixty-five year old female, presented a history of jaundice of three weeks' duration and an 8 pound weight loss. She had no pain. Surgery revealed carcinoma of the head of the pancreas with metastatic lymph nodes and extension into the mesentery of the ileum. Roentgenograms show the brush-like folds on the lower inner duodenal aspect and the loss of plasticity.

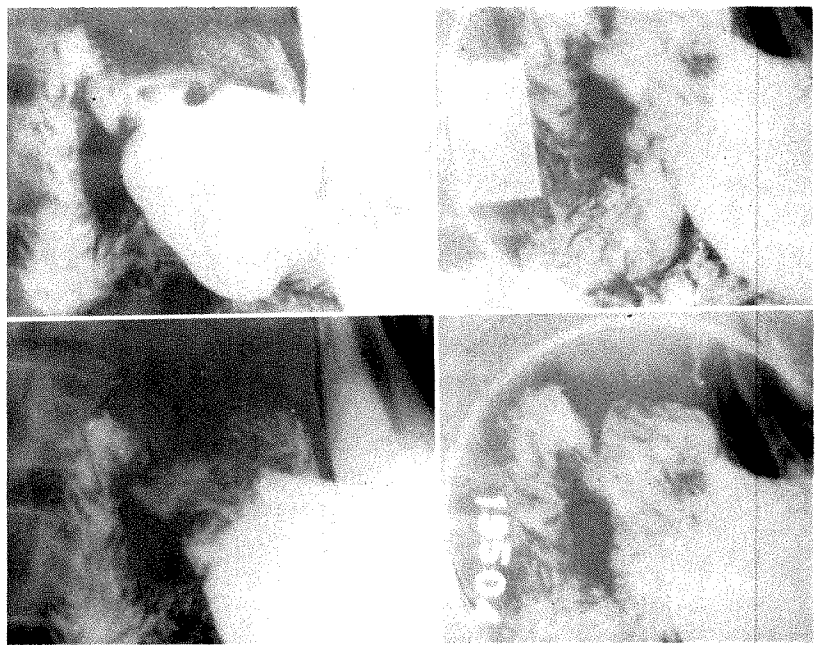


FIG. 4. S.J.M., a forty-five year old male, presented a history of several episodes of pain and nausea during the past forty-six weeks, weight loss, and jaundice of two weeks' duration. Roentgenograms show the two brush-like folds fixed in position at the proximal aspect of the medial second duodenum with flattening of the folds along this area. Autopsy revealed carcinoma of the head of the pancreas with carcinomatosis.

close changes in shape, width, height and direction. Their normal feathery appearance is absent, they assume a brush-like or spike-like configuration (Fig. 3; 4; and 5, *A* and *B*) and occasionally they are flattened and widened (Fig. 6). Some of these folds point constantly into divergent directions, as if fixed in this position by inflammatory or neoplastic changes (Fig. 7, *A* and *B*). Sometimes, the filling of the duodenum is rather fleeting in a thread-like fashion, and an irregular skeletonized ap-

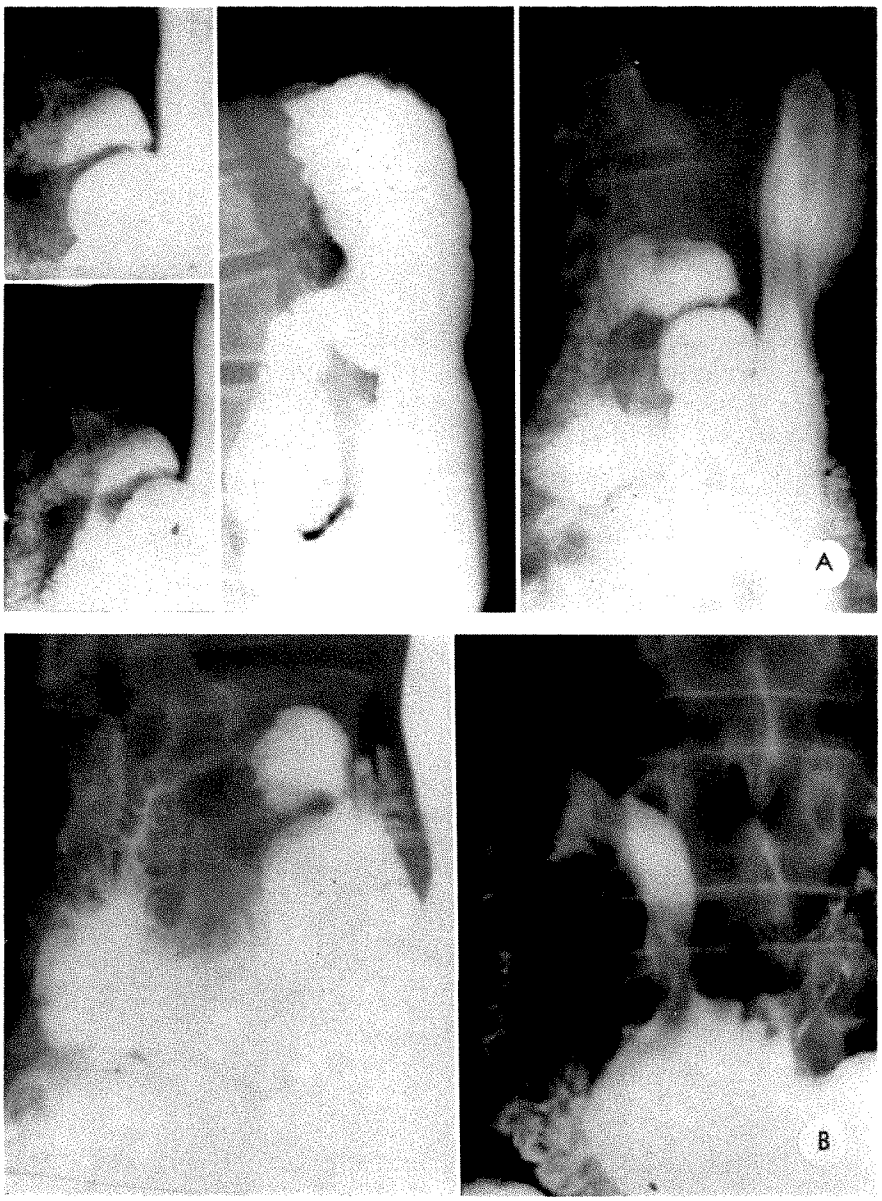


Fig. 5. T.S., a forty-eight year old female, presented a history of abdominal pain, nausea and vomiting of seven months' duration. (*A*) Roentgenograms made at our institution on May 29, 1958 were reported as negative. (*B*) Roentgenograms made in another hospital on June 12, 1959 were also reported as negative. The patient subsequently developed jaundice. Surgery revealed an inoperable carcinoma of the head of the pancreas. Note the triangular fixed fold at the junction of the second and third parts of the duodenum in *A*. Note the double contour of the duodenal loop and the shallow defects on the inner aspect in *B*.

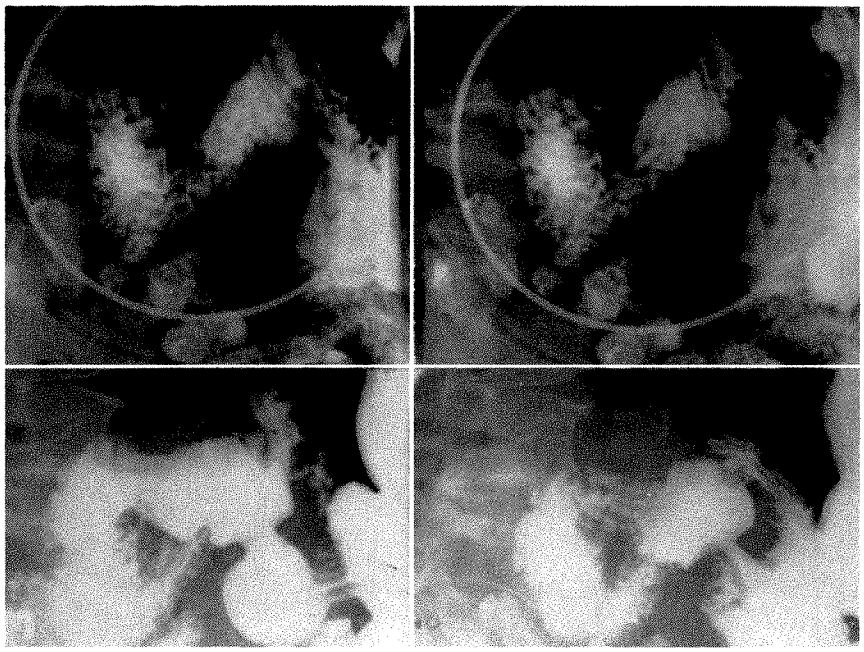


Fig. 6. R.S., a sixty-five year old female, presented a history of pain of five months' duration, which increased in severity on lying down or bending. A mass was felt to the right of the midepigastric region. No jaundice was present and liver function studies were negative. Surgery revealed an adenocarcinoma of the head of the pancreas with metastases to lymph nodes. Roentgenograms show the fixation and flattening of the folds in the lower half of the inner aspect of the second part of the duodenum. This case was not diagnosed roentgenologically.

pearance of thread-like mucosal folds is noted (Fig. 8).

3. Individual folds may become markedly elongated, presenting a pseudodiverticulum of fistula-type formation (Fig. 9). This is probably due to fixation and later necrosis with ulceration produced by the tumor.

4. Duodenal diverticula may be displaced or compressed by the mass (Fig. 10). If a large diverticulum or several diverticula are present, an investigation of the duodenal sweep is very difficult.

5. In the presence of nodularities of the pancreas, indentations on the mesial border of some segments of the duodenal loop develop (Fig. 11; 12; 13; 14, *A* and *B*; and 15, *A* and *B*).

6. With increase in the size of the mass, a concave pressure defect or a double contour of the inner half of the duodenal sweep is noticeable, especially when this area is compressed by a balloon (Fig. 16). The widened common duct produces a flattening of the inner part of the duodenum immediately overlying it (Fig. 17). These double contours are also seen when pathology other than a pancreatico-ampullary carcinoma is present.

7. Post-bulbar impression caused by a widened common duct (Fig. 18 and 19) might occur, and especially in nonjaundiced cases intravenous cholangiography may be helpful. Also, the lateral wall of the duodenum may be compressed by an enlarged gallbladder.

8. In the prone position, the second part of the duodenum is more rigid, drawn mesially, and overlies the bulb and/or antrum, as it is probably attached to the tumor (Fig. 20).

9. Carcinoma of the papilla or ampulla of Vater is sometimes indistinguishable from one arising in the adjacent structures.

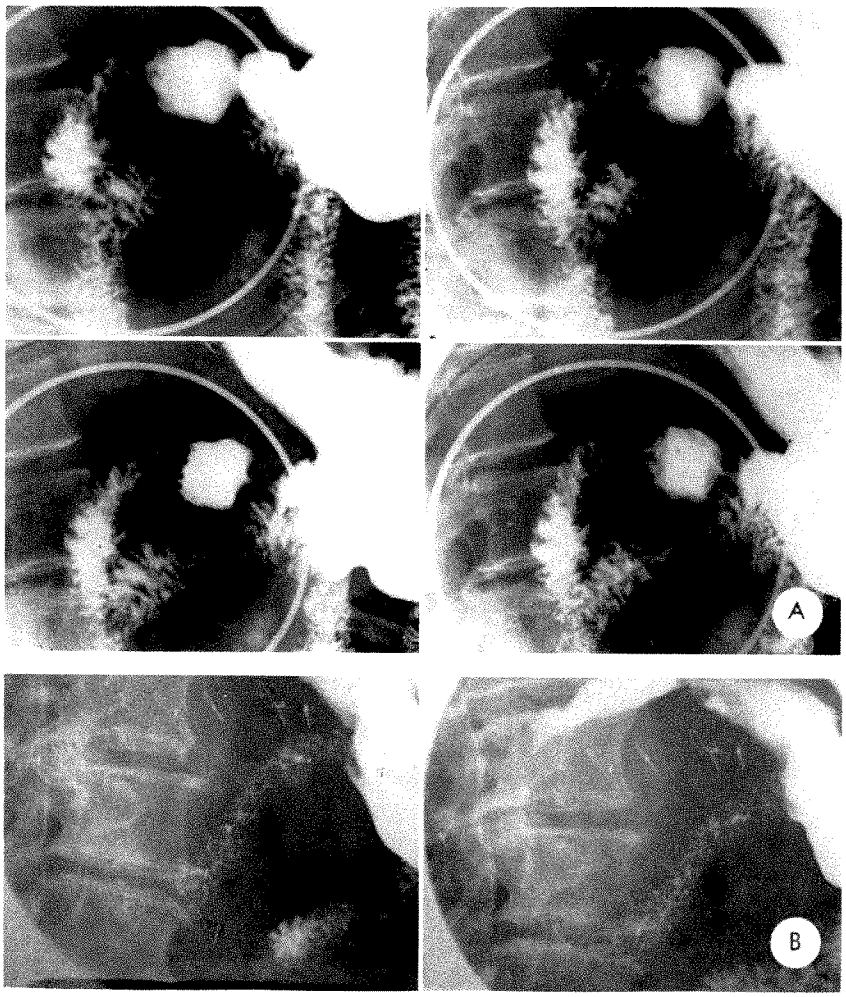


Fig. 7. M.B., a sixty year old female, presented a history of abdominal pain of seven months' duration. There was no jaundice and liver function tests were negative except for slightly high alkaline phosphatase values. Surgery revealed anaplastic carcinoma of the head of the pancreas. (*A*) Roentgenograms made April 5, 1957 show the large spike-like fold with loss of plasticity and the brush-like appearance of the mesial folds of the lower part of the second duodenum. The shallow indentation above the fold is probably caused by the tumor. (*B*) Roentgenograms made November 11, 1957 show the rapid growth of the tumor.

The mucosa is invaded and an irregular filling defect is present (Fig. 21) or an intraluminal mass is noticeable (Fig. 22).

10. With increase in the size of the tumor, the usually sought for and obvious roentgen findings appear; namely, the inverted "3" sign (Fig. 23) (Frostberg[12]), which can also be present in pancreatitis, pad (pressure) effect on the duodenum and antrum with, occasionally, considerable enlargement of the entire loop. Anterior displacement of the duodenal bulb, antrum or body of the stomach is best seen in the right lateral horizontal and left lateral erect positions (Sheinmel and Mednick[28]), and these views are most useful in involvement of the body and/or tail. These tumors of the body and/or tail often produce a change in configuration and depression of the duodenojejunal angle. Newer diagnostic methods like splenoportography and pneumotomography (Moseley[21]), in our



FIG. 8. B.W., a sixty year old male, had had a cholecystostomy for acute cholecystitis with cholelithiasis four months previously. There was no jaundice; alkaline phosphatase was 4.3. Surgery revealed an adenocarcinoma of the head of the pancreas. Roentgenograms show loss of normal configuration of folds at the mesial lower half of the second and third parts of the duodenum. The folds are irritable, flattened in the third part and point in different directions. On most of the roentgenograms a scanty filling of the duodenum with a skeletonized appearance of the folds is present. This case was not reported as a pancreatic carcinoma.

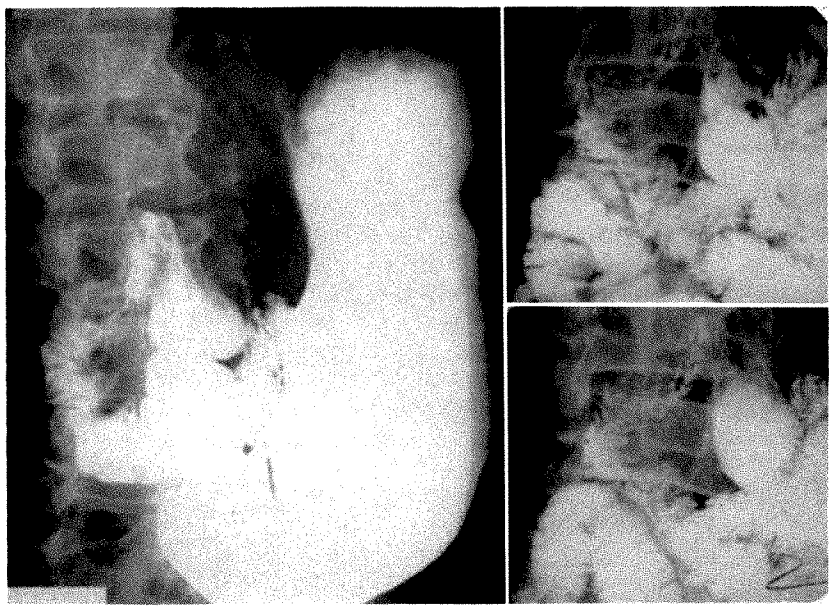


FIG. 9. S.A., a seventy year old male, presented a history of jaundice of six weeks' duration, increasing constipation, an 8 pound weight loss and continuous left upper quadrant pain for the past several months. Surgery revealed carcinoma of the head and body of the pancreas with liver metastases. Roentgenograms show an irregular spike-like fold fixed in position at the junction of the second and third parts of the duodenum with loss of plasticity, irregularity and some flattening of the folds of the third part of the duodenum and pressure by a small mass above.

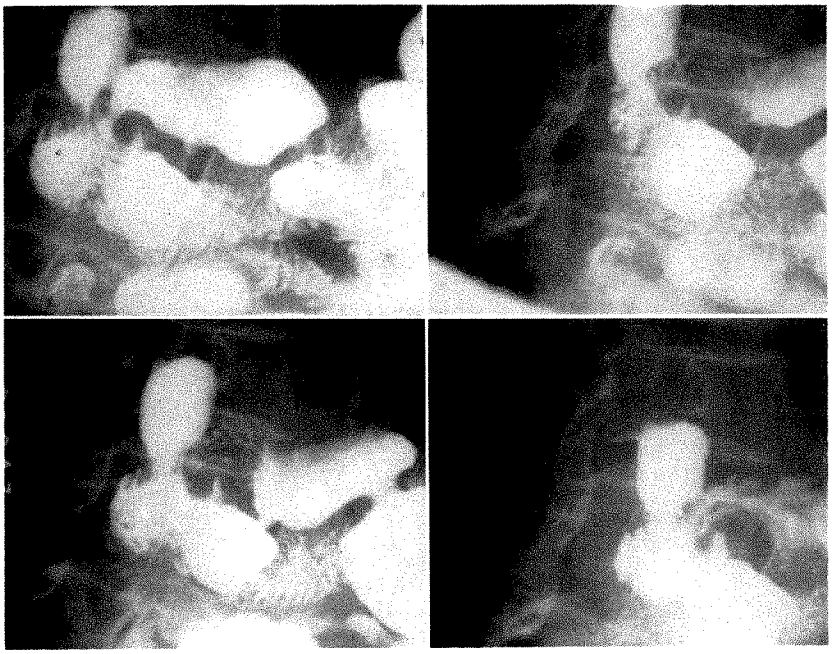


Fig. 10. J.F., a seventy year old female, presented a history of epigastric pain, anorexia and nausea of two months' duration, a 34 pound weight loss since the onset of pain and increasing constipation. She was not jaundiced. An irregular mass was felt in the right upper quadrant. Surgery revealed a lemon-sized carcinoma of the head of the pancreas. Roentgenograms show numerous diverticula, displaced laterally and anteriorly and a triangular fold fixed in position at the junction of the second and third parts of the duodenum. The upper part of the duodenal sweep appears to be compressed by a mass.

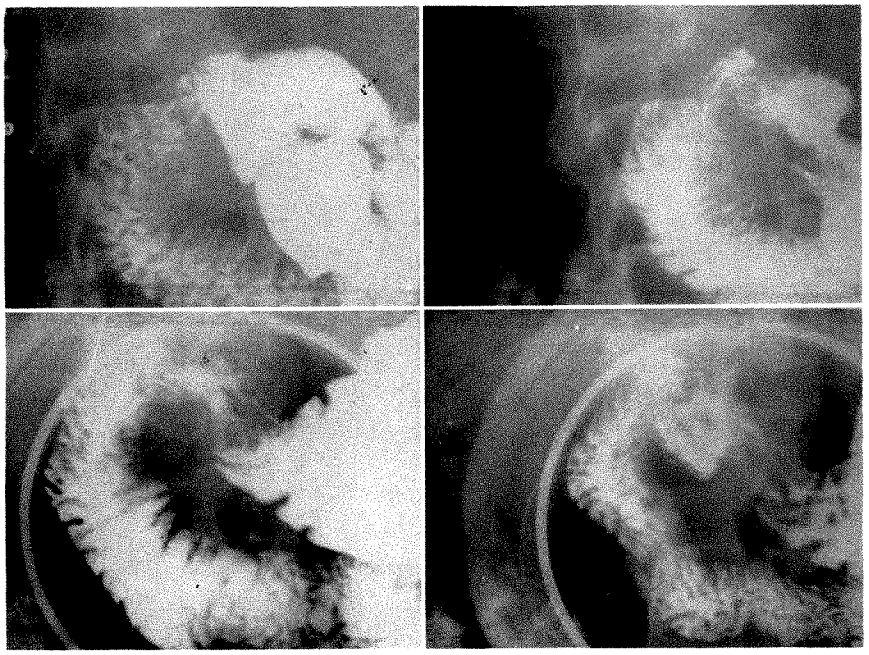


Fig. 11. McC.H., a fifty year old male, presented a history of severe right upper quadrant colicky pain with some weight loss of one and one-half months' duration. Subsequently, he developed jaundice. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the shallow pressure defect caused by a mass and the change in configuration of the folds. A shallow indentation in the post-bulbar area due to an enlarged common duct is also visualized.

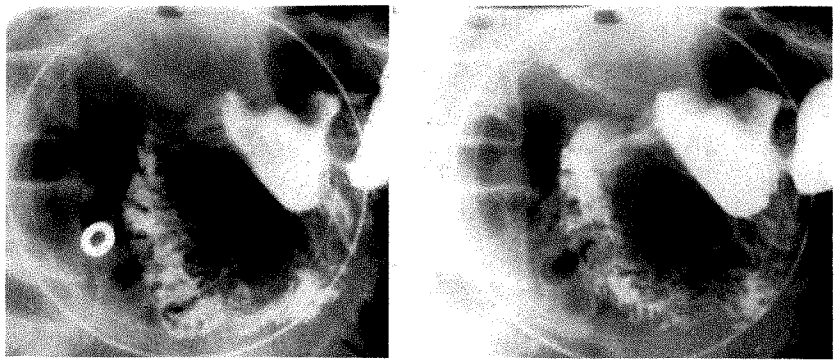


Fig. 12. S.I., an eighty-nine year old male, presented a history of a 12 pound weight loss, anorexia and fullness. Six weeks previously the patient developed jaundice which later subsided and then reappeared. Surgery revealed carcinoma of the head of the pancreas with a markedly enlarged head (10×7 cm. in diameter). Roentgenograms show irregularities of the folds at the inner aspect of the junction of the second and third parts of the duodenum with a change of direction and shallow filling defects.

opinion, disclose tumors in this category when the disease is appreciably advanced. Whenever there is a solitary or concomitant involvement of the head, and in a vast majority this is the case, a routine examination will disclose changes on the inner aspect of the loop without need for more time consuming diagnostic procedures (Fig. 24 and 25). The various stages in development of pancreatic carcinomas and their roentgenologic manifestations are shown on the drawings in Figure 26.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis, other pathologic processes occurring in the head of the pancreas, duodenum, extrahepatic biliary tree and gallbladder or adjacent lymph nodes should be considered.

I. *Pancreas.* A. The most frequent diagnostic problem is caused by pancreatitis which might produce a diffuse enlargement of the head, with compression of the inner second and sometimes third parts (Fig. 27), or localized nodularities with corresponding pressure defects of the duodenum (Fig. 28). Usually, these defects are smooth and the mucosal pattern does not disclose changes noted in pancreatic carcinomas (Fig. 29, *A–C*). Pancreatitis may compress

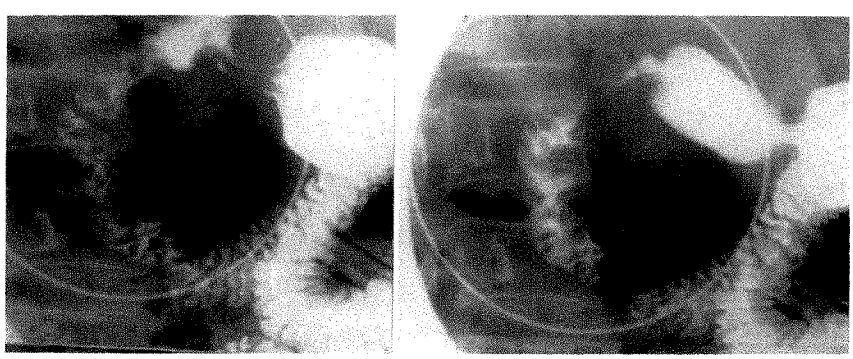


Fig. 13. D.A., a sixty-nine year old diabetic, presented a history of a sudden onset of diarrhea and jaundice of two weeks' duration. During the previous two years, the patient had three episodes of melena, but no pain. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the pseudodiverticulum (?) and irregularities of the inner aspect of the second and third parts of the duodenum with pressure defects and fixation of folds.

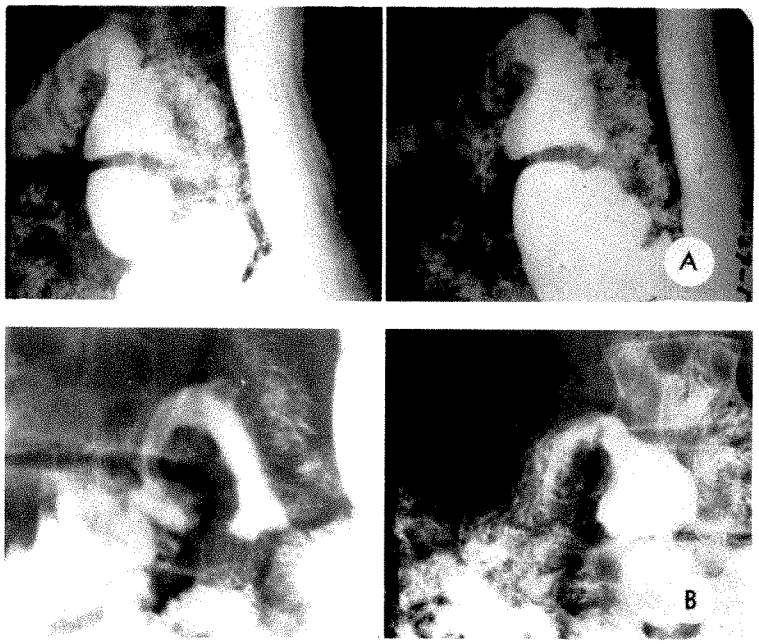


Fig. 14. H.B., a fifty-seven year old female, presented a history of abdominal cramps and nausea of a few years' duration, which three months previously increased in severity and were accompanied by a feeling of weakness, fatigue and subsequently jaundice. Surgery revealed carcinoma, the size of a small plum, located near the upper knee of the duodenum. The pancreas was removed. The patient died two and one-half years later because of metastases. (*A*) Gastrointestinal roentgenograms made October 27, 1953 were negative. (*B*) Gastrointestinal roentgenograms made August 26, 1957 show a mass below the upper knee of the duodenum. A striking difference between *A* and *B* is evident upon comparison.

the common duct and cause jaundice; an enlarged gallbladder may press on the lateral border of the duodenum, a finding which is also noted in the presence of carcinomas of the head of the pancreas (Fig. 30). Occasionally, it is impossible to make a roentgenologic distinction, and the clinical history, signs and symptoms should then be the deciding factors. A swelling of the papilla which is seen in relapsing pancreatitis (papillary sign—Poppel[23]) and possible subsequent development of pressure defects should not be mistaken for the presence of a malignancy.

B. The head of the pancreas may be the site of rare malignant neoplasms, metastases, various cysts (Fig. 31) or benign tumors. If these lesions are small enough to produce early changes on the inner aspect of the duodenal sweep, a differentiation from carcinoma manifested by similar changes might be difficult. Usually benign masses produce smooth defects without any involvement of the mucosa (Fig. 32), whereas metastases produce a roentgenologic pattern which is impossible to distinguish from one caused by a carcinoma of the head of the pancreas (Fig. 33).

C. An annular pancreas produces a quite characteristic pattern of obstruction.

II. *Duodenum.* A. Malignant lesions of the duodenum are usually adenocarcinomas of the scirrhous or papillary type. The scirrhous lesion infiltrates, destroys the mucosa with subsequent rigidity of the wall, shelf-like defects or constriction of the lumen (Fig. 34), and the papillary tumors are intraluminal producing filling defects. Involvement of the duodenum by lymphosarcoma or Hodgkin's disease (Fig. 35) distorts the mucosal pattern, produces a diffuse irregular narrowing of its lumen or a

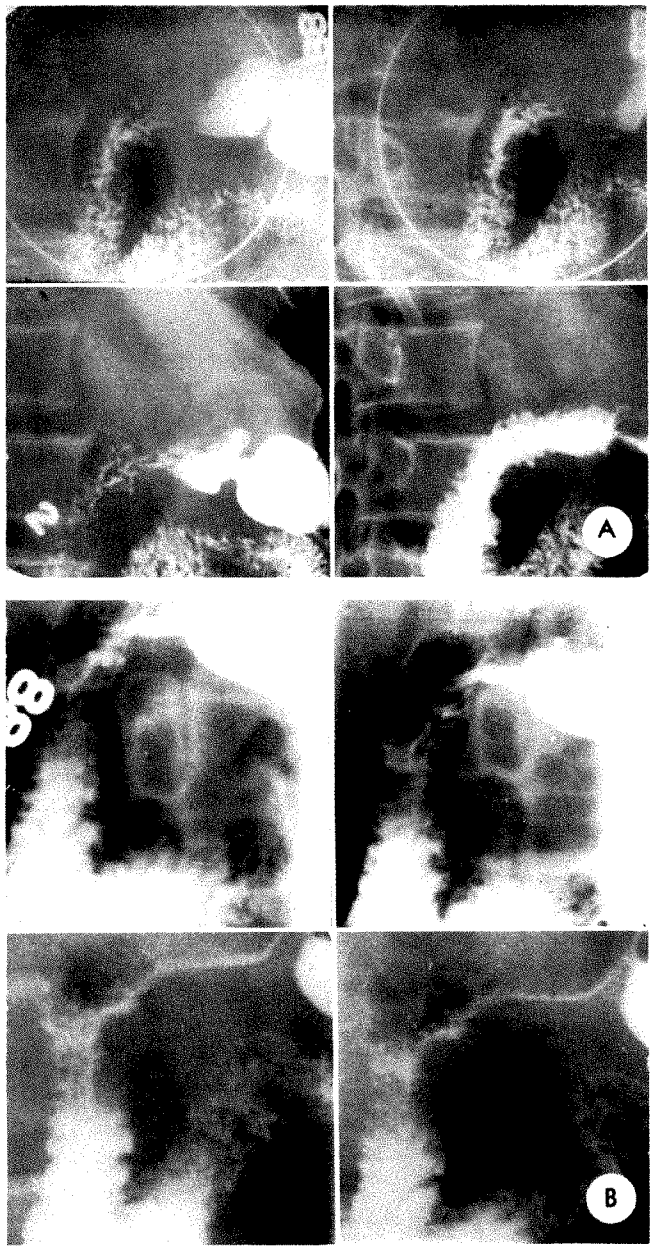


FIG. 15. W.A., a fifty-eight year old male, presented a history of pain in the abdomen, chest and increasing constipation of four months' duration. There was no jaundice. Surgery revealed carcinoma of the head of the pancreas. (*A*) Roentgenograms show the pressure defect caused by a mass in the post-bulbar area, with the folds flattened, fixed and invaded. This type of pressure defect, without involvement of the folds, is also seen in cases of pancreatitis. There is also slight irregularity and fixation of the folds of the distal third part of the duodenum. (*B*) Spot roentgenograms in the erect position. Although the findings may be discernible on the lower roentgenograms taken in a more oblique position, they cannot be visualized on the upper roentgenograms.

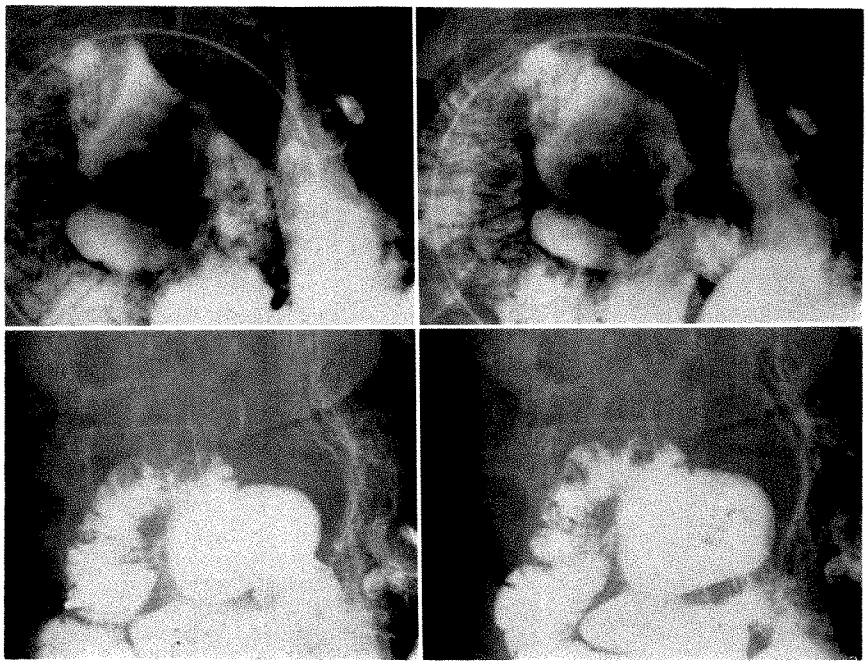


FIG. 16. H.E., an eighty-eight year old female, presented a history of jaundice of two weeks' duration, extensive weight loss and loss of appetite of two months' duration. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the triangular fold projecting mesially and upwards from the lower second duodenal segment and the double contour on pressure studies. Also demonstrated is the post-bulbar pencil-shaped indentation caused by an enlarged common duct and diverticulum with its wall facing the elongated fold slightly compressed.

bag-like appearance when the muscle coat has been replaced by the malignant growth.

B. Post-bulbar ulcers occur probably more often than is realized, and sometimes they are responsible for secondary pancreatitis. A spastic incisura on the wall opposite the ulcer crater is usually present, and this segment of the duodenum is spastic and irritable with coarse mucosal folds. If the ulcer crater is visible, then the diagnostic problem is simplified, but occasionally only an area of irritability and spasticity of the mesial and also lateral

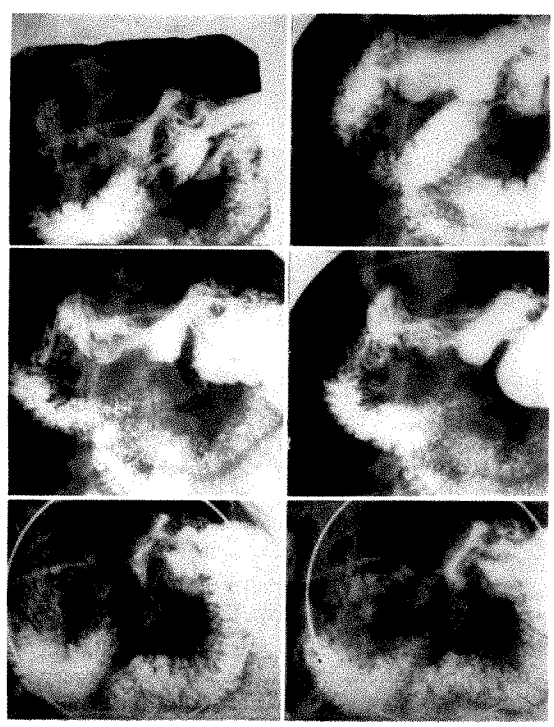


FIG. 17. K.N., a sixty-three year old female, presented a history of nausea of three weeks' duration and jaundice of one week's duration. Surgery revealed carcinoma of the common duct. Roentgenograms show the double contour of the upper inner duodenum with irregular folds.

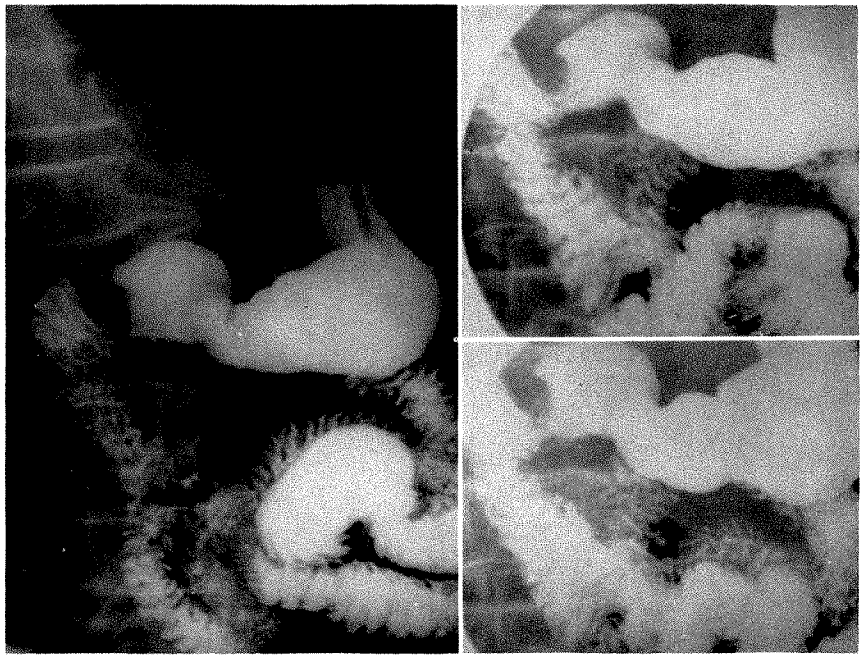


Fig. 18. B.P., an eighty-year old male, presented a history of jaundice of five months' duration. He had no pain but grew progressively weaker and anorexic. Surgery revealed a carcinoma, about 2 cm. in size, of the head of the pancreas or ampulla of Vater. Roentgenograms show the shallow pressure defect and fixation of the folds in the papillary area, also the deep post-bulbar indentation produced by a very large common duct.

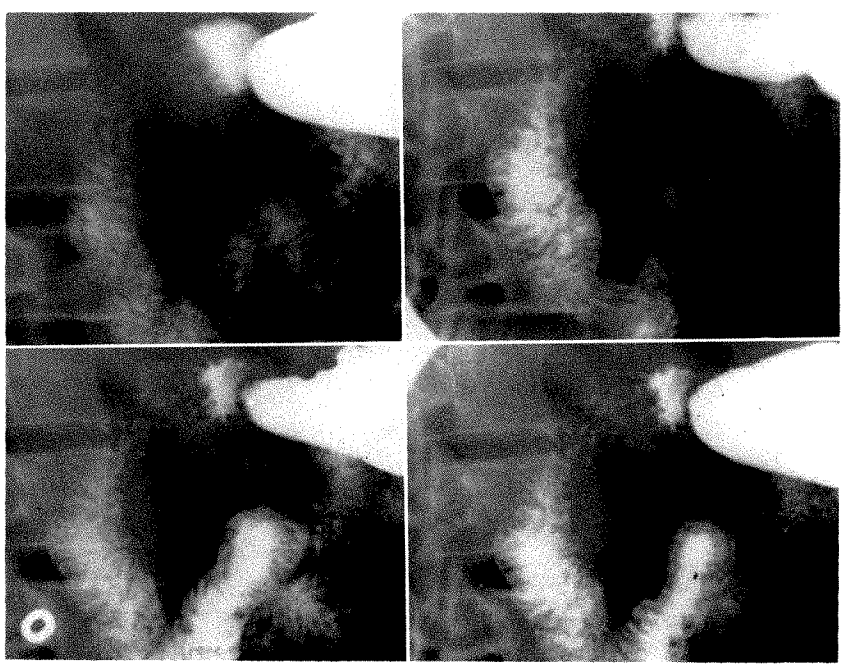


Fig. 19. R.A., a fifty-seven year old male, presented a history of jaundice and weakness of five weeks' duration. During the previous two weeks, he experienced an 8 pound weight loss. At another hospital a diagnosis of infectious hepatitis was made. Surgery revealed a carcinoma of the head of the pancreas. Roentgenograms show the flattening of the folds of the mesial lower second duodenum with a shallow indentation produced by the mass and marked spreading and ironing of the post-bulbar area caused by the enlarged common duct.

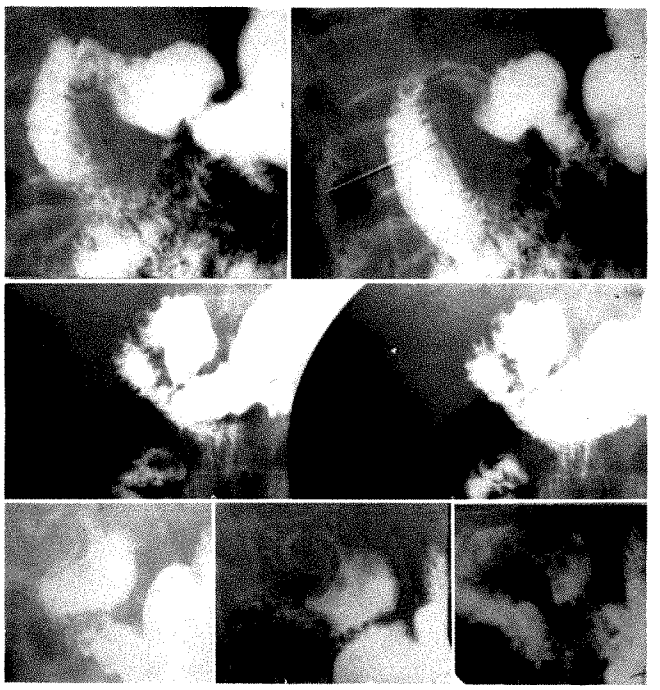


Fig. 20. M.E., a sixty-six year old male, presented a history of first experiencing intermittent epigastric discomfort three months previously. This became continuous, accompanied by a sensation of fullness. He also developed superficial phlebitis of both lower extremities. No jaundice was noted and no masses were felt. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the double contour on the inner aspect of the duodenum with some irregularity of the folds at the lower inner aspect of its second part. In the prone position the mesial direction of the second part of the duodenum is demonstrated.

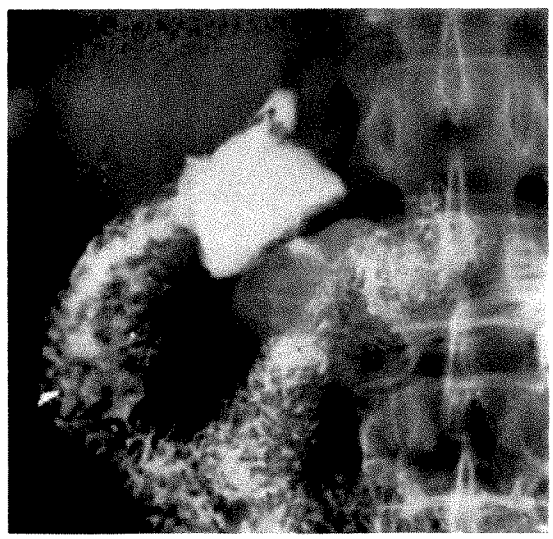


Fig. 21. S.L., a forty-three year old male, presented a history of ulcerative colitis at the age of seventeen. At the age of twenty-two, an ileostomy was performed and at the age of thirty-seven it was closed. In mid-June, 1958, he developed anorexia, progressive jaundice and by August had lost 19 pounds of weight. Surgery revealed tumor in the region of the papilla and the head of the pancreas. A radical pancreatico-duodenectomy was performed. Microscopic examination showed colloid carcinoma of the end of the pancreatic duct in the region of the papilla of Vater. Roentgenogram demonstrates the large irregular filling defect in the region of the papilla with some changes in the folds. (Courtesy of the Department of Radiology, Johns Hopkins Hospital and Medical School.)

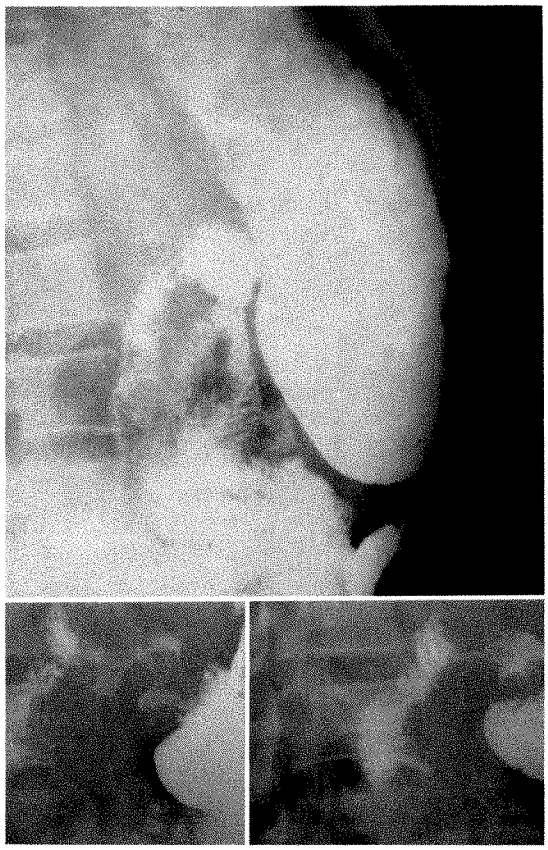


Fig. 22. T.A., a forty-seven year old male, presented a history of jaundice and fatigue of ten days' duration. Surgery revealed a polypoid adenocarcinoma of the ampulla of Vater with involvement of the common bile duct.

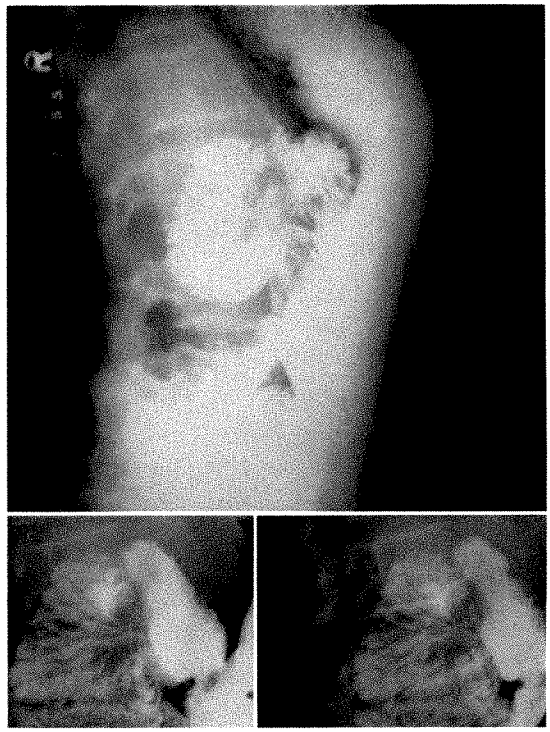


Fig. 23. This sixty-one year old male presented a history of marked weight loss during the previous five months and jaundice of two weeks' duration with bloating and fullness. Surgery revealed a carcinoma of the head of the pancreas with liver metastases. Roentgenograms show the inverted 3 (Frostberg) sign.

walls of the involved segment of the duodenum may be noticeable (Fig. 36). Later, this area may become narrowed due to fibrosis (Fig. 37). Patients with these lesions have a marked tendency to bleed as apparently the ulcer is often located near the pancreatico-duodenal artery which it erodes. Regional enteritis (Berk,[3] McGarity[19]) and tuberculosis (Fig. 38) of the duodenum have been described, but are rare. Usually both borders of the duodenum are involved and some segments irregularly stenosed, with skip areas and string signs.

C. Benign tumors of the duodenum might be due to a leiomyoma (Fig. 39), adenoma, fibroma, lipoma, hemangioma or

Fig. 25. Y.S., a fifty-six year old male, presented a history of sharp mid-thoracic pain radiating across the abdominal wall of five months' duration, increasing constipation of two months' duration, anorexia and fullness. There was no jaundice. Surgery revealed a carcinoma of the entire pancreas, invading the adjacent fundus of the stomach. Roentgenograms show the irregularity with fixation of the folds on the inner aspect of the second duodenum and the presence of a mass on the medial aspect of the fundus.

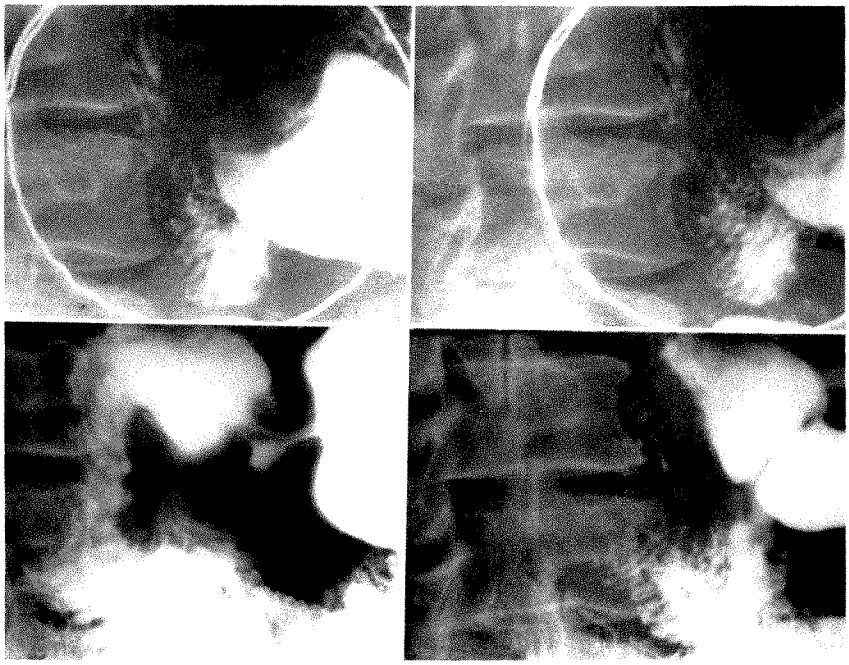
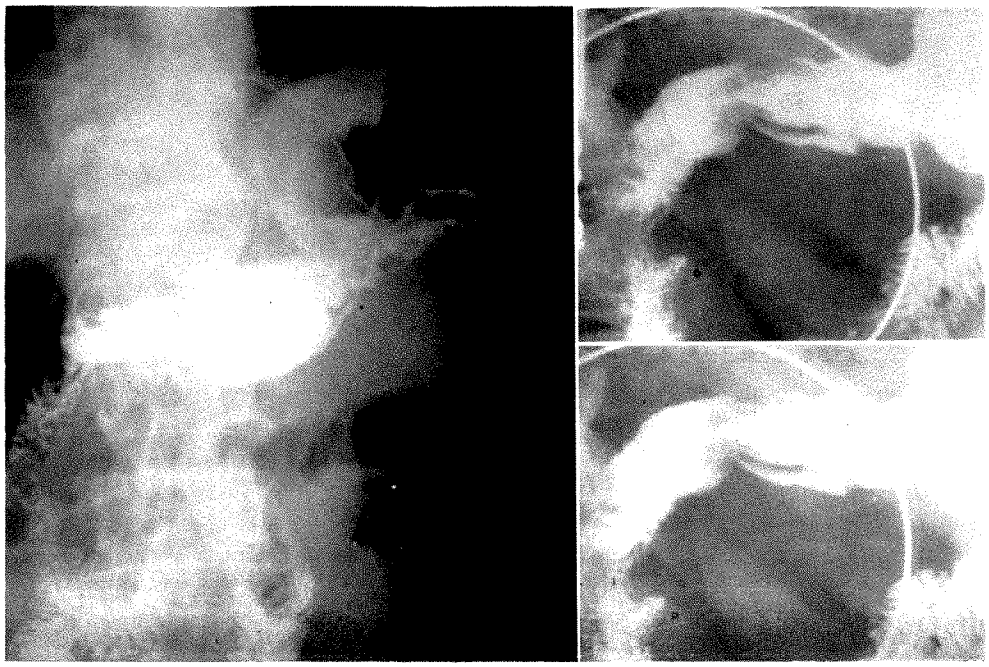


FIG. 24. H.E., a sixty-four year old male, presented a history of diarrhea, a 12 pound weight loss, jaundice of ten days' duration and dull, aching pain in the epigastrium. He had been a heavy drinker until the previous six years and was known to be a diabetic. A firm mass was felt in the left upper quadrant. Surgery revealed a carcinoma of the head and body of the pancreas. Roentgenograms show the irregularity, fixation of folds on the inner aspect of the duodenum and marked irritability of the folds with a skeletonized appearance. The pressure roentgenograms show the antrum to be compressed by the neoplasm. This was also noted in lateral views.



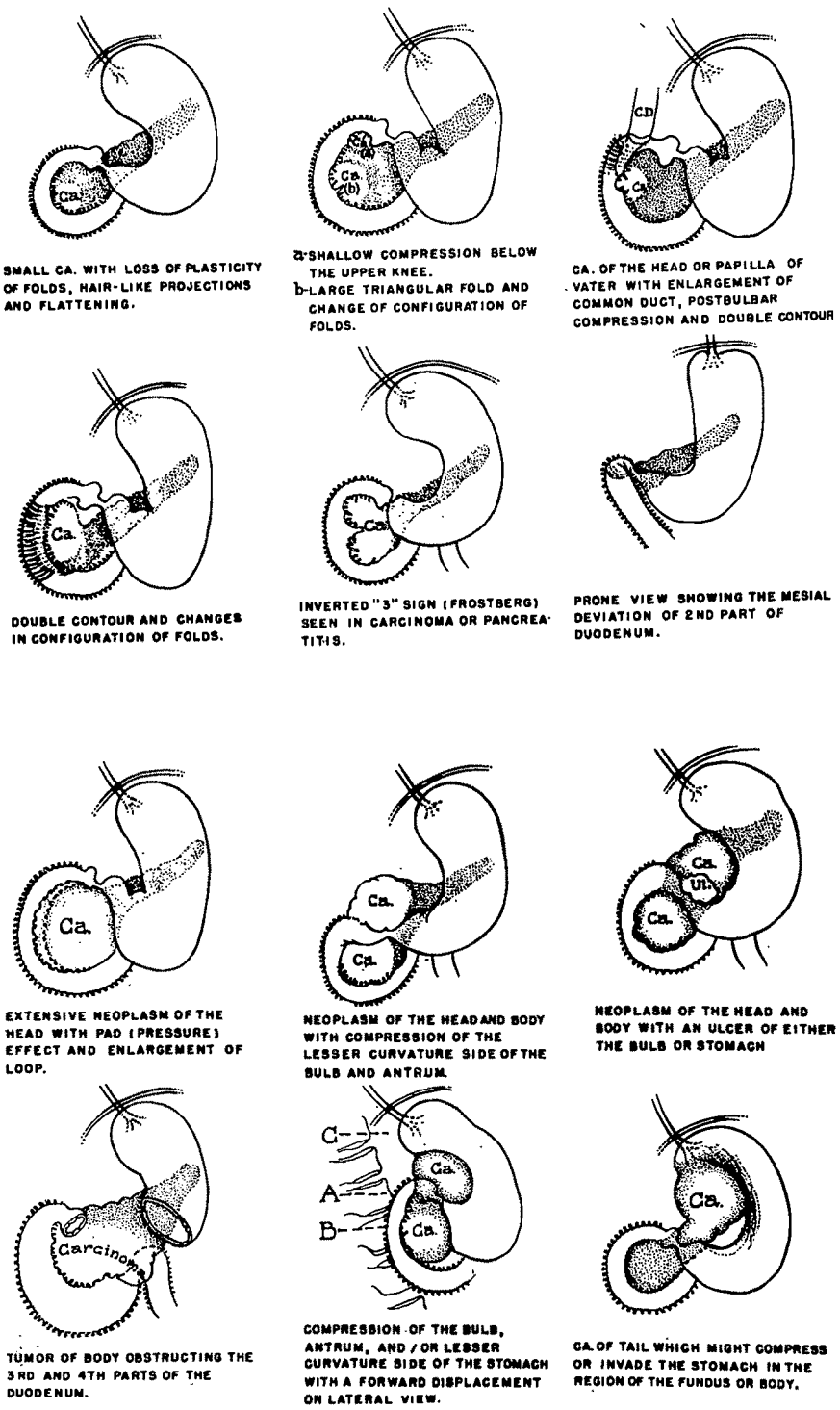


FIG. 26. Drawings showing the different stages in development and roentgenologic manifestations of pancreatic carcinoma.

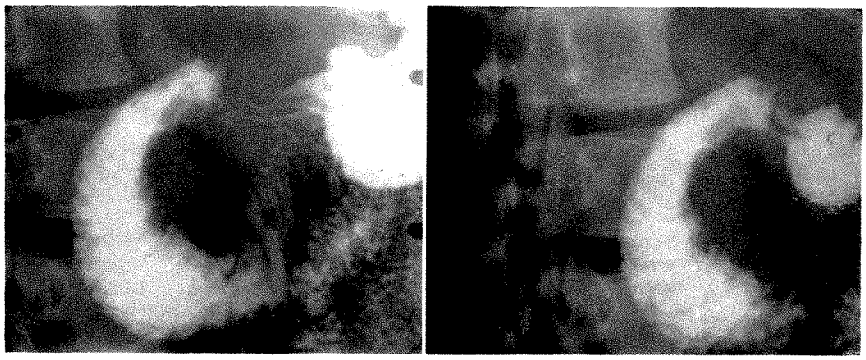


Fig. 27. Ch.M., a twenty-three year old female, presented a history of abdominal pain and vomiting of one and one-half days' duration. At the age of seventeen she began to have sharp mid-epigastric pain radiating to the back. The clinical diagnosis was pancreatitis.

aberrant pancreas. These are usually smooth and produce a discrete filling defect without involvement of the mucosa.

D. A stone lodged within the ampulla of Vater may produce a filling defect with concomitant jaundice (Fig. 40). Ascaris lumbricoides if nestled in the duodenum may show as a filling defect, but none of these roentgen findings could be mistaken for lesions caused by a pancreatico-ampullary neoplasm. Intramural hematomas have a distinctive pattern (Felson and Levin[9]). In the presence of adhesions of the second and third parts, irregularities of the involved duodenal wall may be noticeable (Ochsner[22]).

III. *Extrahepatic biliary tract and gallbladder neoplasms*, if of sufficient size, usually compress the lesser curvature side of the antrum and upper knee and/or descending lateral segment of the duodenum (Fig. 41). Rarely, a large sized tumor of the gallbladder produces a lateral displacement of the descending duodenum that is indistinguishable from a pancreatic carcinoma (McConnell[18]). Choledochal cysts may produce a large pressure defect on the duodenal loop with displacement and also compression of the antrum (Liebner[17]).

IV. *Primary tumors of the liver* usually present no diagnostic problem except that occasionally pancreatic carcinomas may be responsible for large metastases in the liver, or, conversely, liver tumors may metastasize to peripancreatic lymph nodes, and the picture becomes complicated.

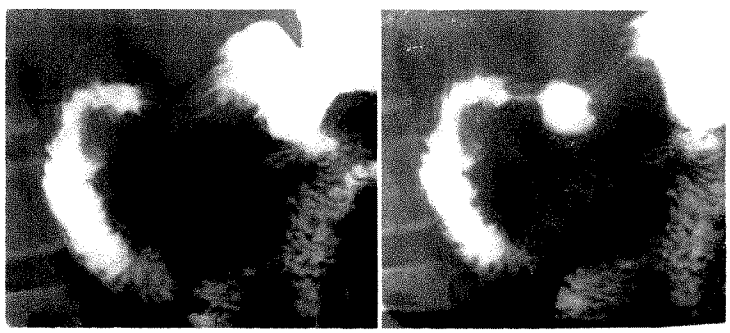


Fig. 28. G.Dm., a thirty-nine year old male, presented a history of intermittent abdominal pain of five years' duration which had recently become severe. The pain started in the left upper quadrant and radiated to the shoulder. The clinical diagnosis was pancreatitis. The roentgenologic pattern of tuberculosis in these cases is sometimes indistinguishable from that of pancreatic carcinoma.

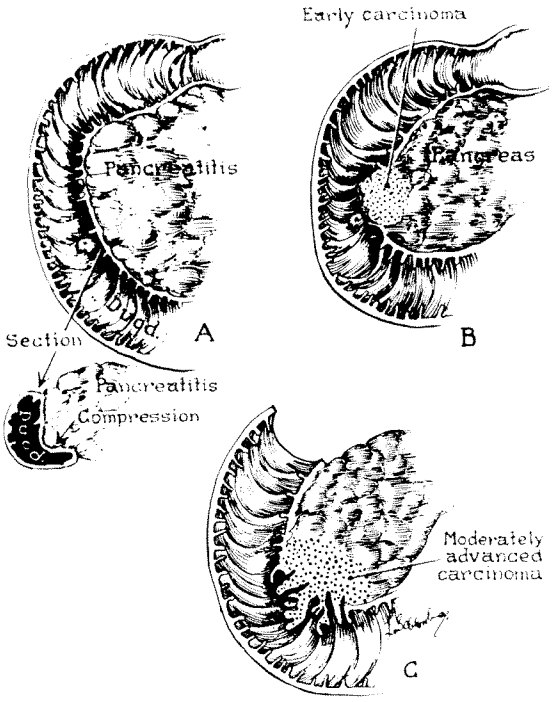


FIG. 29. Drawings show the differential diagnostic patterns of pancreatitis (*A*) and pancreatic carcinoma (*B* and *C*).

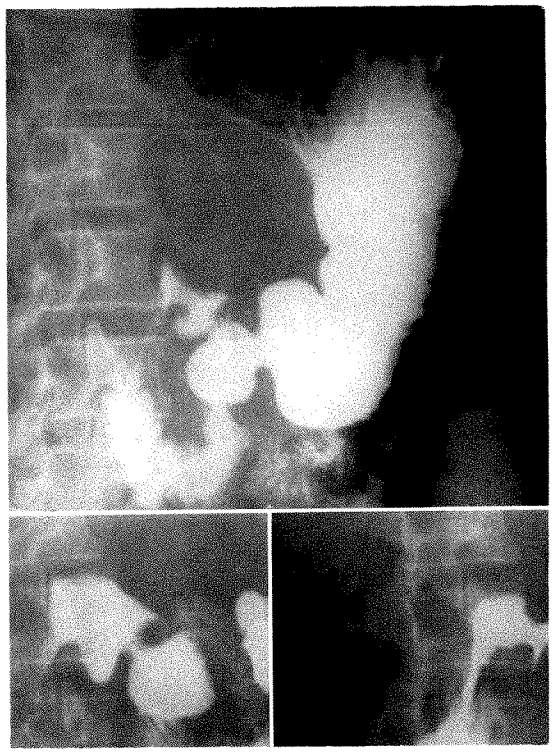


FIG. 30. S.H., a thirty-one year old female, presented a history of sharp right upper quadrant pain of nine days' duration. Surgery revealed chronic cholecystitis and cholelithiasis with chronic pancreatitis.

V. *Retroperitoneal masses* or nodular involvement may occur in primary new growth (sarcoma, lymphoblastoma), metastases or inflammatory processes like tuberculosis or infectious mononucleosis. These retroperitoneal masses at the pancreatic level might produce large pressure defects on the inner aspect of the duodenal loop, or, if nodular involvement is present, multiple scalloped indentations (Poppel[24]). Usually, different surfaces of the duodenum are involved, and the uninvolved area may be completely normal. These masses can be detected on lateral views. In the right prone

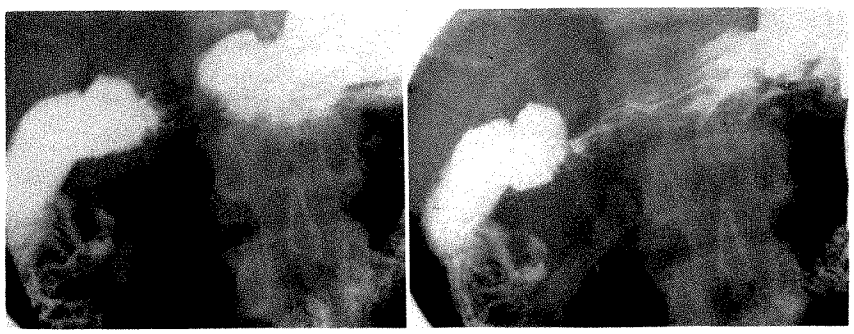


FIG. 31. K.L., a nineteen year old male, presented a history of sharp abdominal pain of eighteen hours' duration and occasional "spasm" of the stomach. Surgery revealed a pancreatic cyst with necrotizing pancreatitis. Roentgenograms show a fist-sized mass in the head of the pancreas.

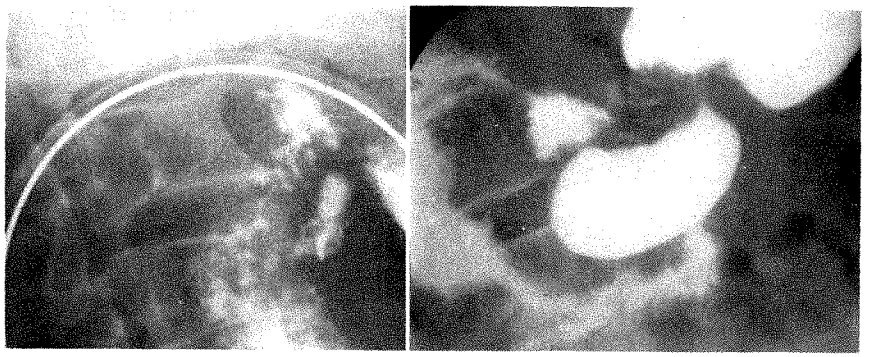


Fig. 32. G.B. presented a history of headache and deep sleep with stertorous respirations. On admission the blood sugar was 57 mg. per cent; after twenty-four hours of fasting, it was 40 mg. per cent. Surgery revealed an islet cell adenoma of the head of the pancreas. Roentgenograms show the compression of the inner aspect of the duodenum below the papillary segment.

anterior oblique position they may imitate tumors of the head of the pancreas, and the differential diagnosis becomes difficult (Fig. 42).

VI. *A large aneurysm of the anterior wall of the abdominal aorta or a hemorrhage around the head of the pancreas* due to a perforation of a vessel may produce a pressure defect on the inner aspect of the sweep (Fig. 43). Also, *lipomatosis* around the head of the pancreas may produce an enlargement of the duodenal loop.

CONCLUSION

1. With recent advances in pancreaticoduodenal surgery an improvement in the long term cure rate in cases of pancreaticoampullary carcinoma is possible, and, therefore, an early diagnosis is of great importance.

2. In the initial stages of the disease the clinical history might not be too distinctive, but some of the symptoms are highly significant and should arouse the suspicion of the radiologist.

3. Early changes on the inner aspect of the duodenal loop can often be detected if a careful examination of this area is conducted, and, thus, a high rate of preoperative diagnostic accuracy can be obtained by roentgenologic procedures.

Department of Radiology
Sinai Hospital
Baltimore 15, Maryland

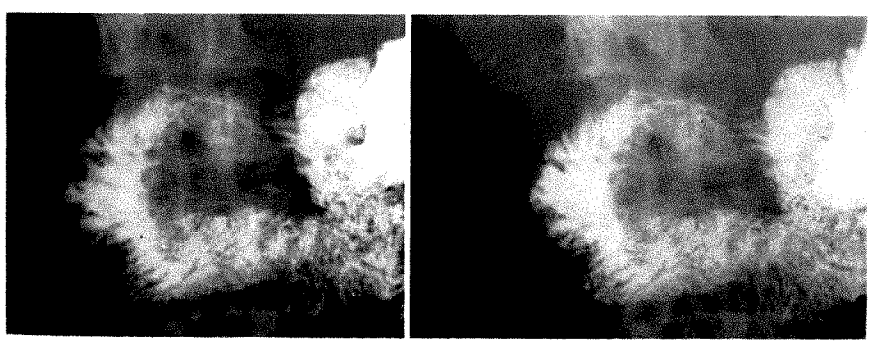


Fig. 33. D.H., a sixty-eight year old male, presented a history of a melanocarcinoma of the scalp with metastases to the cervical lymph nodes three years previously and nausea and a 7 pound weight loss of a few months' duration. Autopsy revealed metastatic melanocarcinoma involving skin, lungs, pancreas, spleen, liver, stomach, adrenal glands and lymph nodes. Roentgenograms show the irregular compression of the inner aspect of the duodenal loop.

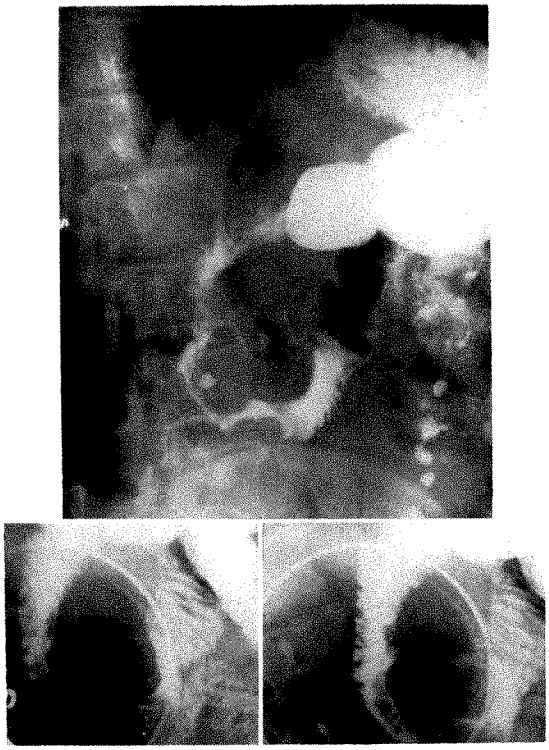


Fig. 34. W.J., a sixty-nine year old white male, presented a history of sharp right upper quadrant pain of four months' duration with constipation for several months. Surgery revealed a fungating carcinoma of the lower second and third parts of the duodenum. Roentgenograms show complete destruction of the mucosa, filling defects, rigidity and a narrowed lumen.



Fig. 35. R.D., a forty-three year old male, presented a history of dull abdominal pain, especially in the right lower quadrant, of eight weeks' duration, with nausea and vomiting and a 32 pound weight loss. Surgery revealed 6 large tumor masses, one in the ileocecal area and 5 along the course of the small intestine with enlarged lymph nodes along the duodenum and intestine. The diagnosis was lymphoblastoma. Roentgenogram shows the irregular narrowing of the entire duodenum; the fourth segment seems to contain a polypoid mass. (Courtesy of the Department of Radiology of the Johns Hopkins Hospital and Medical School.)

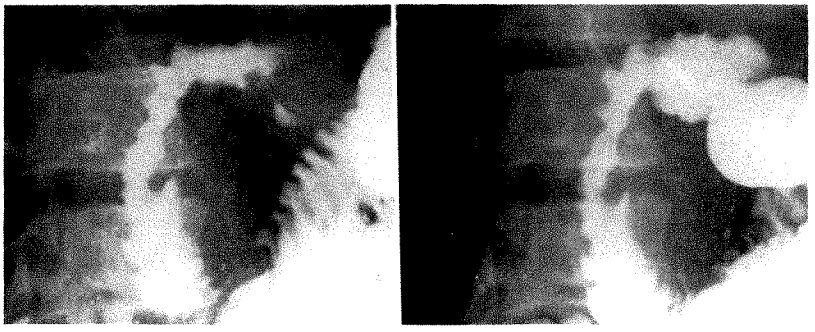


Fig. 36. D.B., a forty-two year old male, presented a history of persistent epigastric pain and occasional melena. Gastrointestinal series prior to admission were reported as negative. Surgery revealed an ulcer of the second part of the duodenum. Roentgenograms show the marked irritability and slight narrowing of the upper half of the second part of the duodenum.

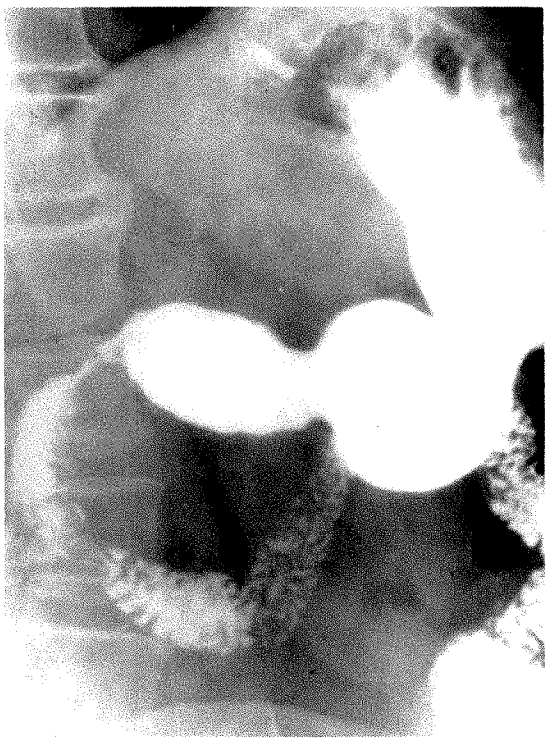


FIG. 37. Roentgenogram shows the constricted postbulbar area and the double contour caused by the enlarged head of the pancreas. In this case surgery revealed ulcer and pancreatitis.



FIG. 38. J.A., a seventeen year old female, presented a history of nine hospital admissions for tuberculosis. The clinical diagnosis was tuberculous enteritis and peritonitis with fistulous tracts between the stomach and small intestine. (Courtesy of the Department of Radiology of the Johns Hopkins Hospital and Medical School.)

REFERENCES

1. BANK, J. Diagnostic problems in cancer of pancreas. *Rocky Mountain M. J.*, 1958, *55*, 37–40.
2. BERK, J. E. Diagnosis of carcinoma of pancreas. *Arch. Int. Med.*, 1941, *68*, 525–559.
3. BERK, M. Regional enteritis involving duodenum; report of three cases. *Gastroenterology*, 1956, *30*, 508–516.
4 BOWDEN, L. Fallibility of pancreatic biopsy. *Ann. Surg.*, 1954, *139*, 403–408.
5. CASE, J. T. Roentgenology of pancreatic disease. Caldwell lecture, 1939. AM. J. ROENTGENOL. & RAD. THERAPY, 1940, *44*, 485–518.

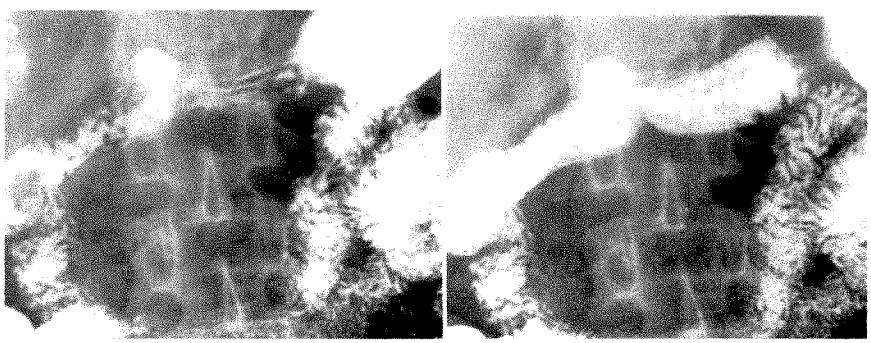


FIG. 39. P.D., a sixty year old male, presented a history of vague abdominal pain of four years' duration and a slightly tender epigastrium. Surgery revealed a tumor, 3 cm. in diameter, in the ampullary area of the duodenum. The pathologic report was leiomyoma.

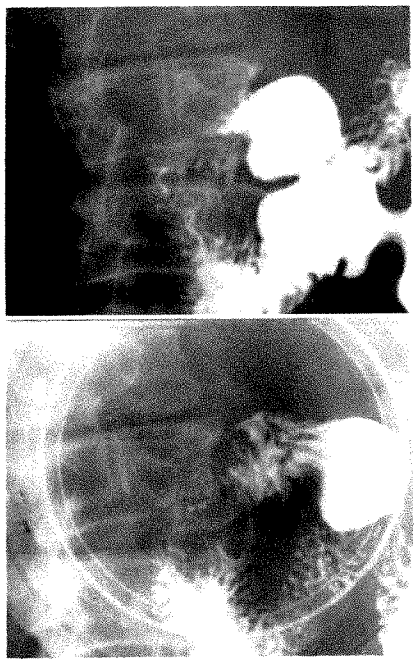
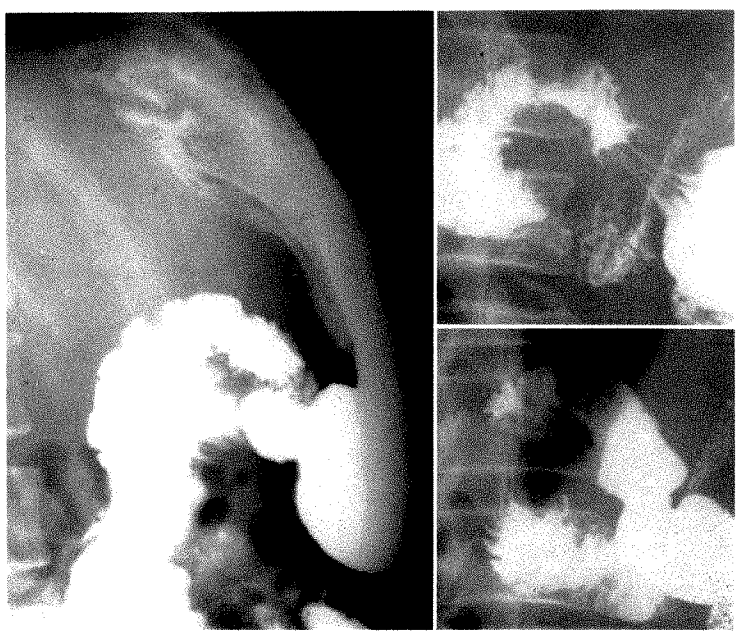


FIG. 40. G.H., a seventy-one year old male, presented a history of abdominal pain of three months' duration and recurrent jaundice. Surgery revealed an oval stone in the papilla with the common duct markedly dilated. Roentgenograms show a defect-like pattern in the ampullary area.



FIG. 41. S.E., a fifty-three year old female, presented a history of the sudden onset of jaundice and mild mid-thoracic and epigastric pain of two weeks' duration. Surgery revealed a carcinoma of the gallbladder. Roentgenograms show the compression of the lateral aspect of the duodenum.



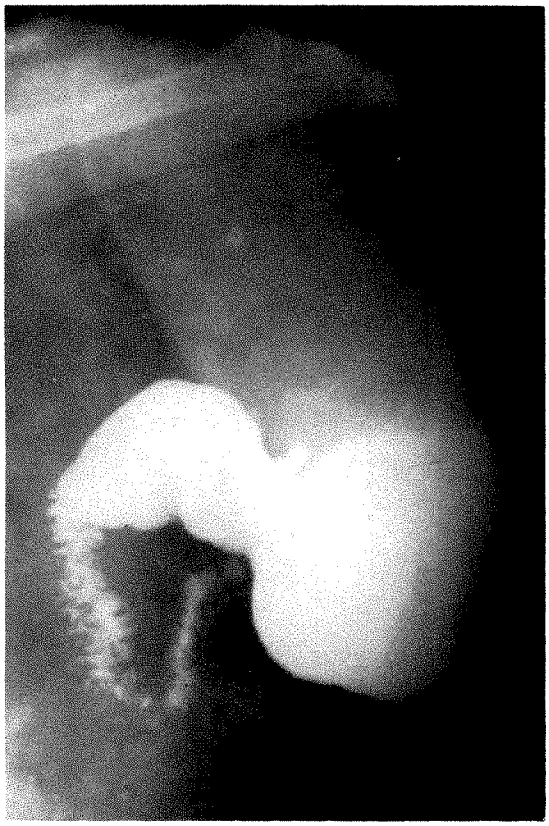


FIG. 43. M.E., a seventy-six year old male, was admitted as an emergency patient with sharp laminating pain in the left upper quadrant. He died twelve days later. At autopsy a mass of clotted blood surrounded the head of the pancreas and the second part of the duodenum, a result of perforation of the superior mesenteric artery. Roentgenogram shows the compression of the duodenum. During fluoroscopy the obstruction of the second duodenum was quite impressive.

6. CATTELL, R. B., WARREN, K. W., and AU, F. T. C. Periampullary carcinomas; diagnosis and surgical management. *S. Clin. North America*, 1959, *39*, 781–798.
7. CLIFFTON, E. E. Carcinoma of pancreas. *Am. J. Med.*, 1956, *21*, 760–780.
8. DENNIS, C., and VARCO, R. L. Survival for more than five years after pancreato-duodenectomy for cancers of ampulla and pancreatic head. *Surgery*, 1956, *39*, 92–106.
9. FELSON, B., and LEVIN, E. J. Intramural hematoma of duodenum. *Radiology*, 1954, *63*, 823–831.
10. FRANTZ, V. K. Tumors of the Pancreas. Armed Forces Institute of Pathology, Section VII, Fascicles 27 and 28.
11. FRASER, J. Surgical treatment of obstructive jaundice in pancreatic disease. *Brit. J. Surg.*, 1938, *26*, 393–411.
12. FROSTBERG, N. Characteristic duodenal deformity in cases of different kinds of perivaterial enlargement of pancreas. *Acta radiol.*, 1938, *19*, 164–173.
13. GULLICK, H. D. Carcinoma of pancreas; review and critical study of 100 cases. *Medicine*, 1959, *38*, 47–84.
14. HODES, P. J., PENDERGRASS, E. P., and WINSTON, N. J. Pancreatic, ductal, and vaterian neoplasms: their roentgen manifestations. *Radiology*, 1954, *62*, 1–15.
15. JOHNSON, T. A., and KALSER, M. H. Pancreas; contributions of clinical interest. *Gastroenterology*, 1959, *36*, 295–332.
16. KLINE, J. R., and CULVER, G. J. Roentgen findings in primary duodenal and paraduodenal malignant lesions. AM. J. ROENTGENOL. & RAD. THERAPY, 1947, *58*, 425–438.
17. LIEBNER, E. J. Roentgenographic study of congenital choledochal cysts; pre- and postoperative analysis of five cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 950–960.
18. MCCONNELL, F. Malignant neoplasm of gall bladder: roentgenological diagnosis. *Radiology*, 1957, *69*, 720–725.
19. MCGARITY, W. C. Regional enteritis of duodenum. *Surg., Gynec. & Obst.*, 1957, *105*, 203–209.
20. MILLBOURN, E. Experiences of radical pancreatic surgery in cases of carcinoma of head of pancreas or papilla of Vater. *Acta chir. scandinav.*, 1958, *116*, 1–11.
21. MOSELEY, R. D., JR. Diagnosis of tumors of pancreas with aid of pneumoretroperitoneal pancreatography and splenoportography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 967–977.
22. OCHSNER, H. C. Roentgenologic diagnosis of periduodenal and extraduodenal lesions. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *45*, 718–723.
23. POPPEL, M. H. Roentgen manifestations of re-

FIG. 42. F.T., a forty-five year old male, presented a history of loss of weight, anorexia and abdominal pain. A hard mass was palpable in the left upper quadrant. Surgery revealed an undifferentiated malignant lymphoma of the retroperitoneal tissues with involvement of the mesenteric and pancreatic lymph nodes.

lapsing pancreatitis. *Radiology*, 1954, *62*, 514–521.

24. Poppel, M. H. Roentgen Manifestations of Pancreatic Disease. Charles C Thomas, Publisher, Springfield, Ill., 1951, p. 389.
25. Porter, M. R. Carcinoma of pancreaticoduodenal area; operability and choice of procedure. *Ann. Surg.*, 1958, *148*, 711–724.
26. Rendich, R. A., Poppel, M. H., and Cove, A. M. Roentgen diagnosis of space-occupying lesions in region of head of pancreas. *Radiology*, 1942, *38*, 47–53.
27. Schiff, L. Absence of palpable liver—sign of value in excluding obstructive jaundice due to pancreatic cancer. *Gastroenterology*, 1957, *32*, 1143–1144.
28. Sheinmel, A., and Mednick, E. A. Roentgen diagnosis of upper abdominal retroperitoneal space-occupying lesions. Am. J. Roentgenol. & Rad. Therapy, 1951, *65*, 77–92.
29. Waugh, J. M., and Gilbertson, R. G. Radical resection of head of pancreas and duodenum for malignant lesions. *S. Clin. North America*, 1957, *37*, 965–979.
30. Wise, R. E., and Johnston, D. O. Roentgenologic diagnosis of malignancy of pancreas. *S. Clin. North America*, 1956, *36*, 699–706.
31. Zintel, H. A., Rhoads, J. E., and Helwig, J., Jr. Results of subtotal pancreatectomy in treatment of carcinoma of pancreas. *West. J. Surg., Gynec. & Obst.*, 1959, *67*, 293–299.

VOL. 86, NO. 1

VISCERAL "AIR LUMENOGRAM"

By S. MARTIN BRONSON, M.D.,* *and* SIMON KRANTZ, M.D.†
ATLANTA, GEORGIA

A CAREFUL study of the radiolucent visceral air shadows on the plain roentgenogram or other roentgenograms of the abdomen will occasionally reveal organic lesions of the gastrointestinal tract. This paper reports 9 cases in which signs suggestive of gastric peptic ulcer disease, gangrenous appendicitis, and neoplasm of the stomach and colon were evident on the plain roentgenograms of the abdomen. The significant changes were seen retrospectively except in 3 cases—a gastric lymphoma (Case V), a gangrenous appendicitis (Case IV), and a gastric ulcer (Case I). However, with more careful examination of the roentgenograms, the diagnosis might have been suggested by the preliminary and ancillary abdominal studies (*e.g.*, the cholecystograms, excretory urograms, etc.). We have not attempted to determine from our material the percentage of cases in which such findings will be visualized on the plain roentgenogram of the abdomen; it is obvious that it would be extremely difficult to obtain such figures.

The visceral gas pattern or outline is routinely studied in those cases presenting clinical evidence of intestinal obstruction, perforation of a viscus, or an intra-abdominal mass. Roentgenograms obtained incident to the study of the urinary collecting system, the lumbosacral spine, etc. usually are not adequately scrutinized for the intestinal gas pattern.

As early as 1917, Carman and Miller[1] noted variations in the size or the configuration of the stomach fundus gas bubble. They pointed out that the normally regular and symmetric outline of the gas bubble might be broken by a splenic or hepatic tumor. They also noted that an intrinsic tumor, involving the cardia and, usually, the lesser curvature side, produced an opaque deformity in this aspect of the gastric bubble. Kirklin[4] later stressed the importance of this manifestation and, in an analysis of 68 cases of tumor of the cardia of the stomach, he noted that a mass projecting into, or producing a defect in, the gas bubble was seen on routine chest roentgenograms in about 50 per cent of the cases.

The stomach gas bubble normally presents a hemispheric radiolucent outline below the left hemidiaphragm. A polypoid new growth projecting into the lumen of the stomach will produce a deformity of this gas bubble.

We are presenting 9 cases in which there was a consistent and persistent distortion of the configuration of a gas filled viscus on two or more roentgenograms, and have designated the term "air lumenogram" as a descriptive label for such visceral changes observed on the plain abdominal roentgenograms. These cases are submitted to stimulate a wider interest in the closer evaluation of the abdominal gas pattern.

GASTRIC PEPTIC ULCER

In 3 cases of peptic ulcer disease, the ulcer was clearly outlined by air on a plain roentgenogram of the abdomen. The lesions measured from 1.4 to 3.0 cm. in their greatest diameter, and were proven by surgical exploration.

In Case I (Fig. 1, *A* and *B*), the ulcer was visualized on a plain roentgenogram of the abdomen obtained eight days prior to performing an upper gastrointestinal series. In Case II (Fig. 2, *A* and *B*), the "air lumenogram" of the ulcer was visualized on a roentgenogram made one month following the upper gastrointestinal studies.

* Present address: S. Martin Bronson, M.D., Department of Radiology, Carter County Memorial Hospital, Elizabethton, Tennessee.

† Chief, Radiology Service, Veterans Administration Hospital, and Associate Professor of Radiology, Emory University School of Medicine, Atlanta, Georgia.

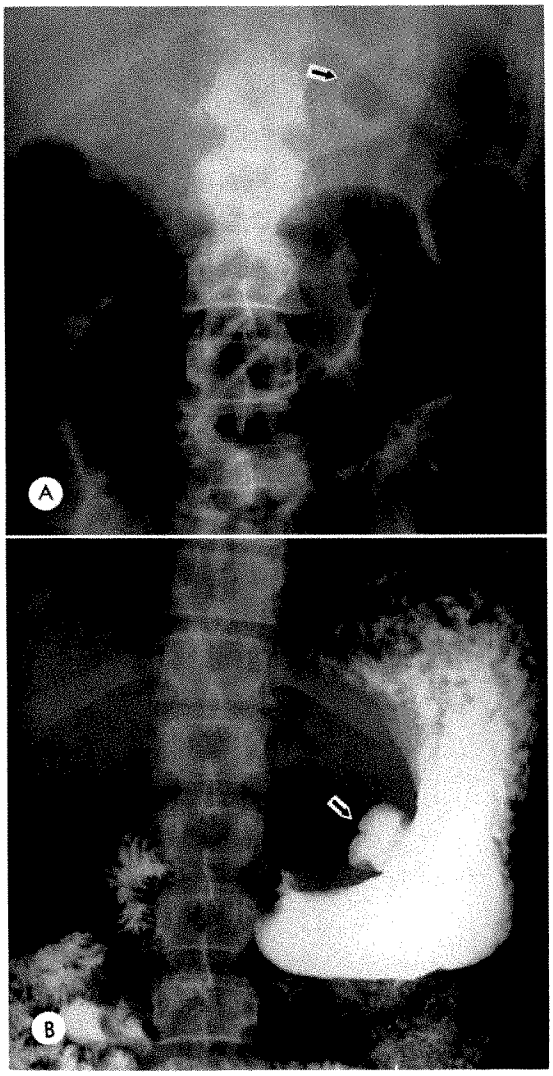


FIG. 1. Case I. (*A*) The large gastric ulcer niche (arrow) on the lesser curvature is sharply delineated, outlined by air. Even the meniscus of mucosal thickening can be discerned. The patient, a forty-two year old male alcoholic, admitted February 21, 1957, was referred from the emergency room in an intoxicated condition and with a history of gastrointestinal bleeding for several hours. This roentgenogram was obtained subsequent to the upper gastrointestinal series. (Courtesy of Dr. Marvin Silverstein, Atlanta, Georgia.) (*B*) Upper gastrointestinal roentgenogram reveals a large, penetrating, peptic gastric ulcer (arrow) and a duodenal ulcer (the latter better demonstrated on other roentgenograms). Surgery substantiated the roentgen findings.

In Case III (Fig. 3, *A*, *B* and *C*), the ulcer was visualized on a barium enema roentgenogram made seven days subsequent to an upper gastrointestinal series.

The configuration of the air shadow of the ulcer and its position, as observed in these cases, are believed to be sufficiently characteristic for at least a presumptive diagnosis.

GANGRENOUS APPENDICITIS

Fischer[2] recently presented 1 case of a dilated, fixed, gas filled appendix with an air fluid level indicative of a gangrenous, nonperforated appendix. It has been well documented that the normal appendix may contain air only intermittently.[3] A non-

FIG. 3. Case III. (*A*) Upper gastrointestinal roentgenogram, February 18, 1959. The recurrent ulcer of the lesser curvature is in precisely the same location as the gastric ulcer seen in 1956. On surgical exploration, a 3×2 cm. benign peptic ulcer was found on the lesser curvature. (*B*) Barium enema study, February 25, 1959. Note the round, sharply defined radiolucent area in the left upper abdominal quadrant, lying medial to the splenic flexure and cephalad to the air-filled gastric antrum (arrow). (*C*) Line drawing of *B* showing air outlining the gastric ulcer.

This forty-four year old male had initial symptoms of abdominal pain in 1942. In 1956, roentgen examination revealed a gastric ulcer of the lesser curvature, 10 mm. in diameter, which, with subsequent medical management, showed healing roentgenographically. Six months prior to the above upper gastrointestinal studies, he experienced recurrence of severe abdominal pain, frequent vomiting, and a thirty pound weight loss.

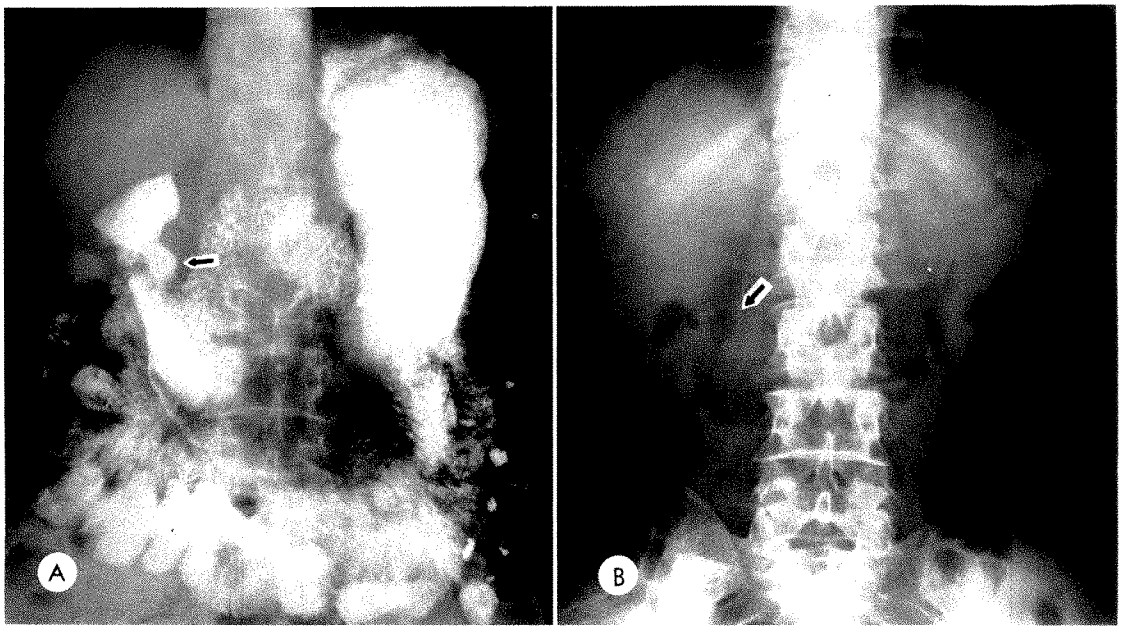
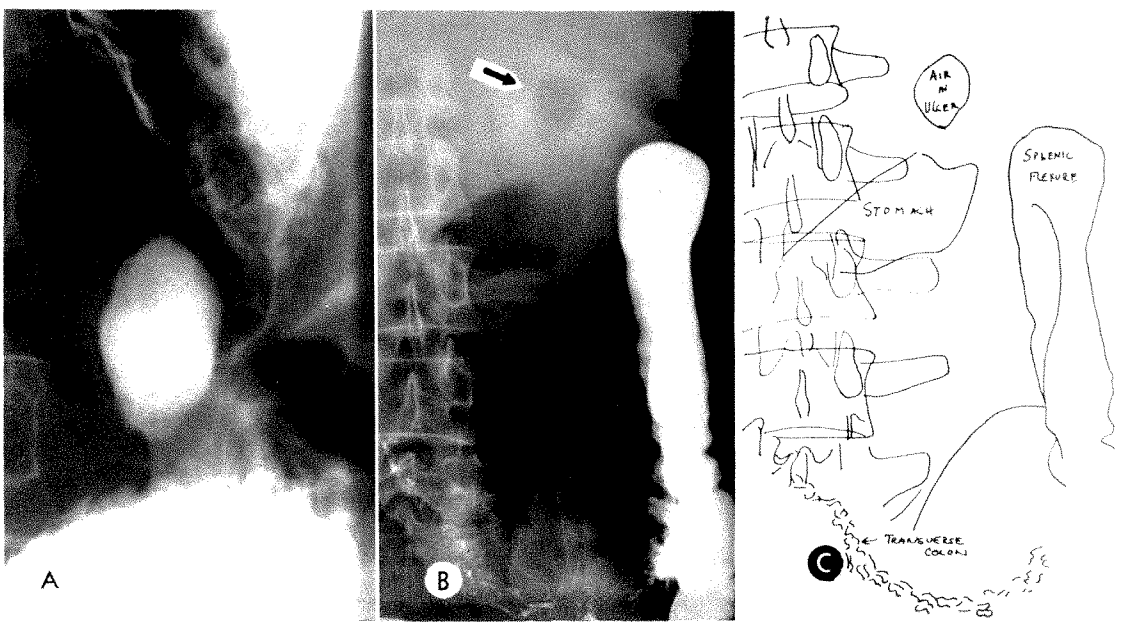


Fig. 2. Case 11. (*A*) Upper gastrointestinal roentgenogram, May 25, 1959, demonstrates a large ulcer crater (arrow) of the prepyloric area. This was also observed on two previous gastrointestinal examinations with the oral administration of barium. (*B*) Roentgenogram, taken on readmission, June 26, 1959, when surgery was proposed for partial obstruction and intractable pain secondary to pyloric ulcer, shows an ulcer crater of the pylorus outlined by air (arrow). Surgical exploration revealed a 1.4 cm. by 0.2 cm. deep prepyloric ulcer along the lesser curvature of the stomach. There was no evidence of neoplasm on pathologic examination. (Courtesy of Dr. Joseph Chang, Emory University Hospital, Atlanta, Georgia.)



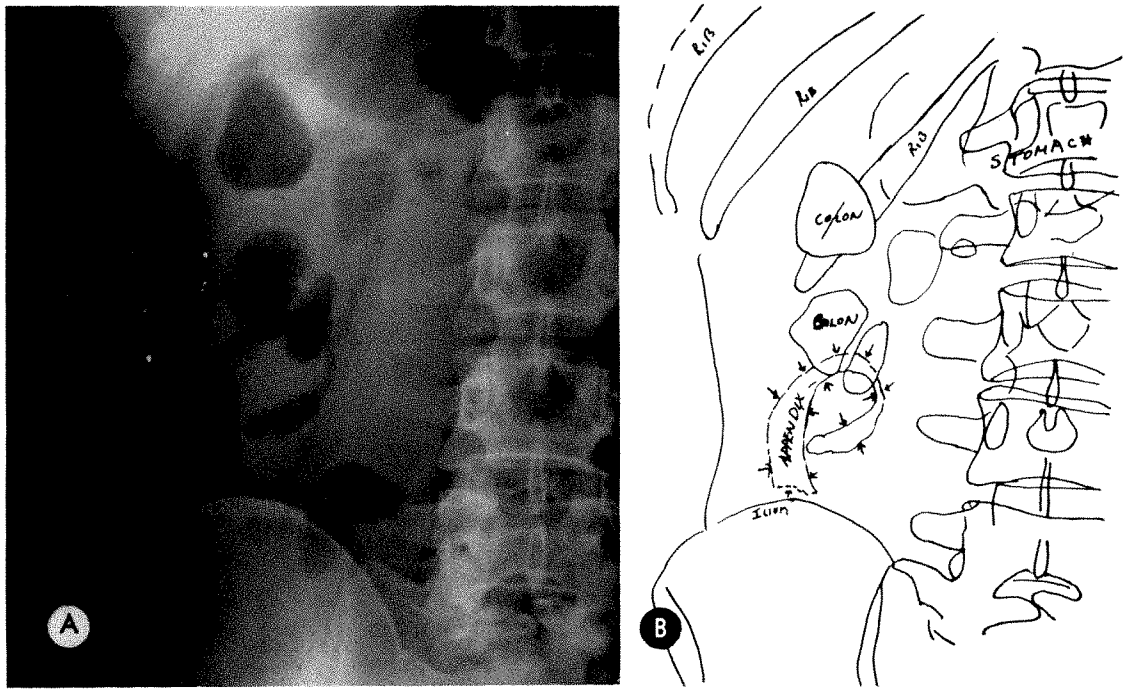


FIG. 4. Case IV. (*A*) Flat roentgenogram of the abdomen, January 3, 1959. Air outlining the lumen of the appendix is well visualized. (*B*) Line drawing of *A*.

The patient, a thirty-two year old white male and a diabetic for thirteen years, had onset of abdominal pain three days prior to the date of this study with marked tenderness over the right lower quadrant on light palpation. At appendectomy, performed on January 3, 1959, a purulent gangrenous perforated appendix was found with a large, adjacent, inflammatory mass lying retrocecally along the lateral peritoneal wall. Culture of the appendix revealed aerobacteria and numerous gram positive spores; the types were not determined on subsequent cultures.

pathologic appendix in the subhepatic area or in the presence of distention of the right colon, as in paralytic ileus, may contain gas without a fluid level.

Case IV, a thirty-two year old white diabetic male, had a gangrenous, perforated appendicitis. The appendiceal lumen on the flat roentgenogram of the abdomen was sharply outlined by air (Fig. 4, *A* and *B*). Unfortunately, an upright roentgenogram of the abdomen was not obtained so that the presence or absence of fluid and the degree of immobility of the appendix was not roentgenographically determined. Surgical exploration on the same day as the roentgen examination revealed a purulent, gangrenous, perforated appendix, adherent to an adjacent inflammatory mass. This would indicate that the air filled appendix can be seen in gangrenous appendicitis, even though perforation may have taken place.

NEOPLASM OF THE STOMACH

A case of Hodgkin's disease involving the cardia, fundus, and lower esophagus with invasion of the left hemidiaphragm showed a deformity of the cardia with a mass projecting into the stomach air bubble (Case V, Fig. 5, *A* and *B*). This was clearly visualized on the posteroanterior chest roentgenogram. The deformity was substantiated by an upper gastrointestinal series and by surgical exploration.

In another instance (Case VI, Fig. 6, *A*, *B* and *C*), an excretory urogram revealed a persistent abnormal deformity of the stomach outlined by air; this lesion was

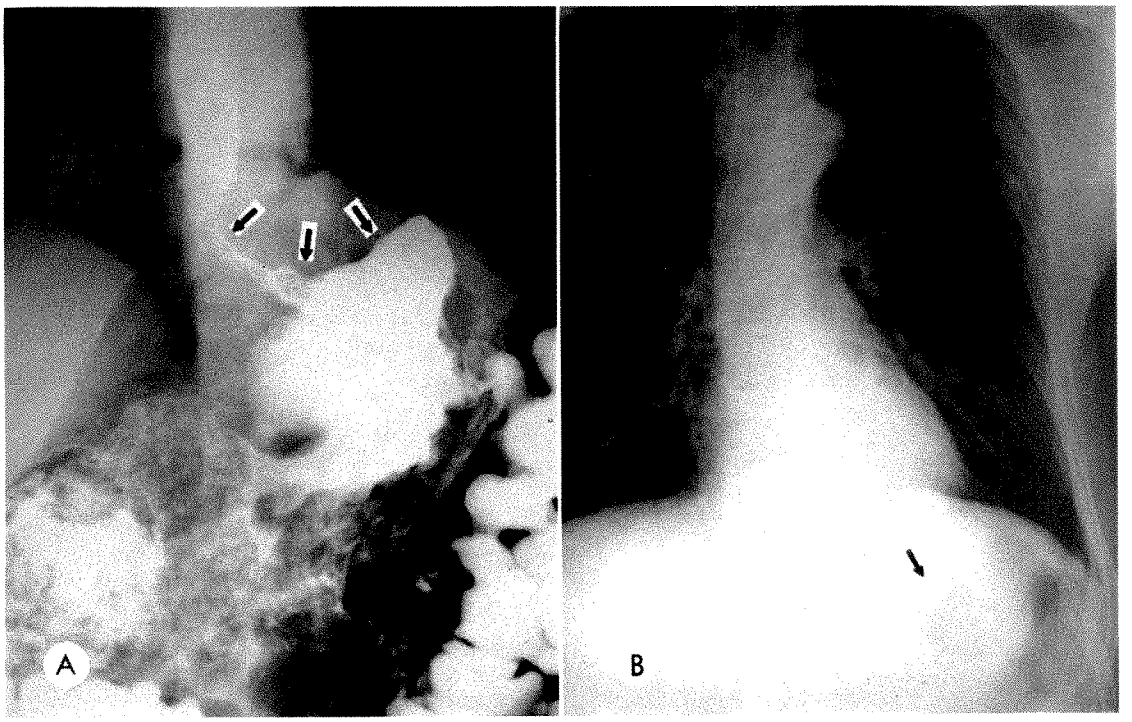


Fig. 5. Case v. (*A*) Upper gastrointestinal roentgenogram, February 12, 1959, of a fifty year old white male who had a history of weight loss, shows an extensive organic lesion (arrows) involving the cardia and fundus of the stomach and extending above the diaphragm to involve the distal esophagus. Surgical exploration revealed Hodgkin's disease of the stomach and distal esophagus with invasion of the diaphragm. (*B*) Posteroanterior chest roentgenogram, February 6, 1959, shows the neoplasm deforming the cardia and fundus of the stomach, with air outlining the inferior margin of the tumor mass.

confirmed by barium study. On abdominal exploration, an inoperable adenocarcinoma of the stomach was found.

NEOPLASTIC LESIONS OF THE COLON

Three cases are presented in which the abdominal roentgenograms demonstrated malignant lesions of the colon. In Case VII (Fig. 7, *A*, *B* and *C*), an excretory urogram revealed the typical finding of a lesion involving the ascending colon. A similar pattern was demonstrated on barium enema examination performed two days later. An operable carcinoma of the ascending colon was found at surgery.

The second case (Case VIII, Fig. 8, *A*, *B* and *C*), a sixty-nine year old patient, was treated for genitourinary sypmtoms in December, 1957. On plain roentgenograms of the abdomen an irregular tapering of the proximal splenic flexure was observed at the site of a carcinoma. A roentgen diagnosis of this defect was not made until some time later, August 21, 1958, by means of a barium enema examination. A more careful study of the original plain roentgenograms would have led to an earlier diagnosis.

In the third patient (Case IX, Fig. 9, *A*, *B* and *C*), a plain roentgenogram of the abdomen obtained on April 26, 1959, revealed an intraluminal polypoid mass in the descending colon. This was definitively visualized, of course, on barium enema studies the following day.

SUMMARY

A gastric ulcer niche was demonstrated

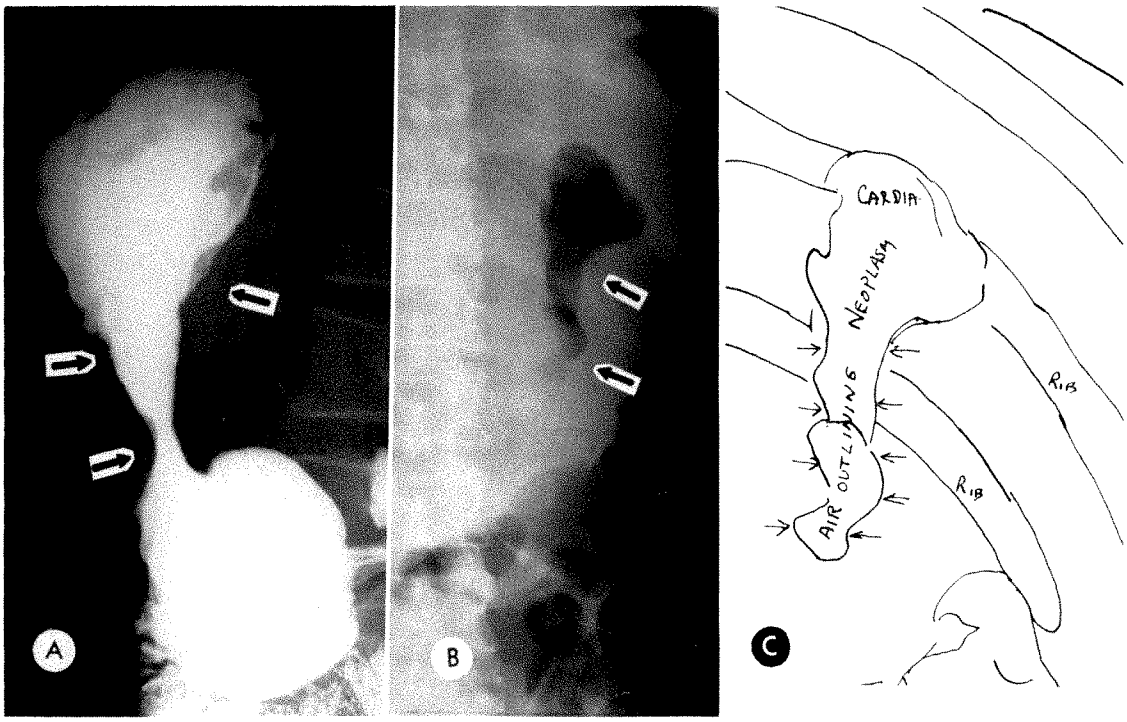


FIG. 6. Case VI. (*A*) Upper gastrointestinal roentgenogram, July 29, 1958. An annular, stenotic, rigid, organic process (arrows), apparently a neoplasm, is involving the greater and lesser curvature of the stomach. At abdominal exploration, an inoperable adenocarcinoma of the stomach metastatic to the omentum was found. (*B*) Flat roentgenogram of the abdomen, August 6, 1958, obtained during exploratory urography. Essentially the same stereotyped narrowed body of the stomach is seen outlined by air. (*C*) Line drawing of *B*.

This fifty-nine year old white male was well until four weeks prior to admission, July 25, 1958, when he developed an epigastric, gnawing pain and a slight, retrosternal, burning sensation.

in 3 cases on the plain roentgenograms of the abdomen; these ulcers were clearly defined.

A patient with gangrenous appendicitis revealed an appendiceal lumen sharply outlined by air. It is of interest that the fixed appendix was found to be perforated at the time of surgical exploration.

Two neoplasms of the stomach were visualized on roentgenograms of the chest and abdomen, respectively.

Three neoplasms involving the colon, (the ascending colon, splenic flexure, and descending colon) were clearly outlined by air on plain roentgenograms of the abdomen.

In only 3 of the above 9 cases (Hodgkin's disease of the stomach, an air-filled gangrenous appendix, and a gastric ulcer), was the diagnosis made from the plain roentgenogram. Nevertheless, the air outline or visceral "lumenogram" in each instance presented an appearance characteristic enough to have suggested the diagnosis.

Definitive gastrointestinal studies with contrast media are essential to confirm the impressions gained from the "air lumenograms." The extreme variability of the mucosal pattern or configuration of the stomach does not allow a presumptive diagnosis based only on a single plain roentgenogram of the abdomen. However, if a typically pathologic gastric pattern, not only of the fundus but also of the body, is visualized on the plain roentgenogram, it may be suggestive of the diagnosis.

Retrospective examination of the roentgenograms of several patients with neo-

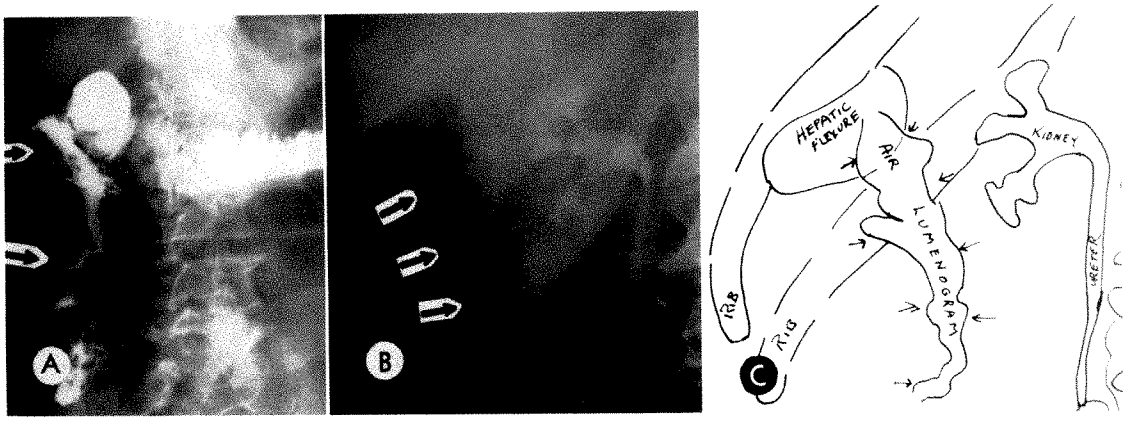


FIG. 7. Case VII. (*A*) Barium enema study, July 11, 1958, indicates an organic lesion (arrows) of the ascending colon measuring 8.5 cm. in length. This seventy year old Negro male four months prior to admission had noted right abdominal discomfort ("gas pains"). He presented with evident, moderate, recent, weight loss, anemia, and a palpable mass in the right upper quadrant. (*B*) Excretory urogram, July 9, 1958, shows displacement of the renal shadow toward the midline. The rigid, typical, long curvilinear contours outlined by air (arrows) represent the neoplasm of the ascending colon. (*C*) Line drawing of *B*. The arrows indicate the narrowed stenotic lumen of the ascending colon involved by neoplasm. At operation, a 9 cm. carcinoma of the ascending colon was found immediately adjacent to the hepatic flexure, with invasion of the posterior abdominal wall, the psoas muscles, and the vena cava.

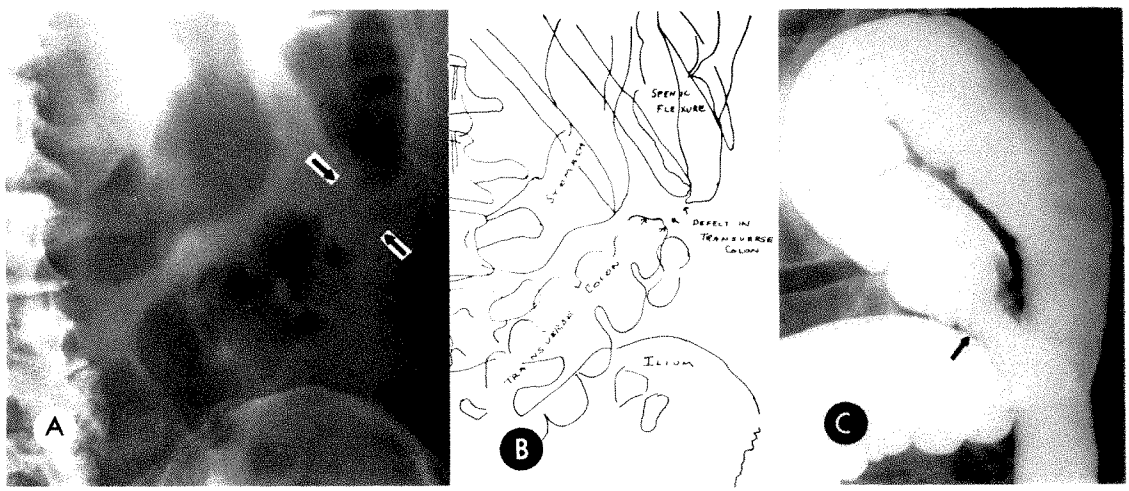


FIG. 8. Case VIII. This sixty-nine year old Negro male was on the Genitourinary Service from December, 1957, to March, 1958, for benign prostatic hypertrophy treated by transurethral resection. (*A*) Plain roentgenogram of the abdomen obtained during excretory urography, December 17, 1957. Arrows indicate an irregular tapering of the proximal splenic flexure caused by a carcinoma which was not diagnosed until later. Note also the absence of air at the site of the carcinoma, with moderate distention of the colon proximal to the lesion. (*B*) Line drawing of *A*. (*C*) Barium enema study, August 21, 1958, after the patient was readmitted for anorexia, marked weight loss, lower mid-abdominal pain and constipation. An adenocarcinoma of the transverse colon just proximal to the splenic flexure, measuring 4×5 cm., was found on surgery.

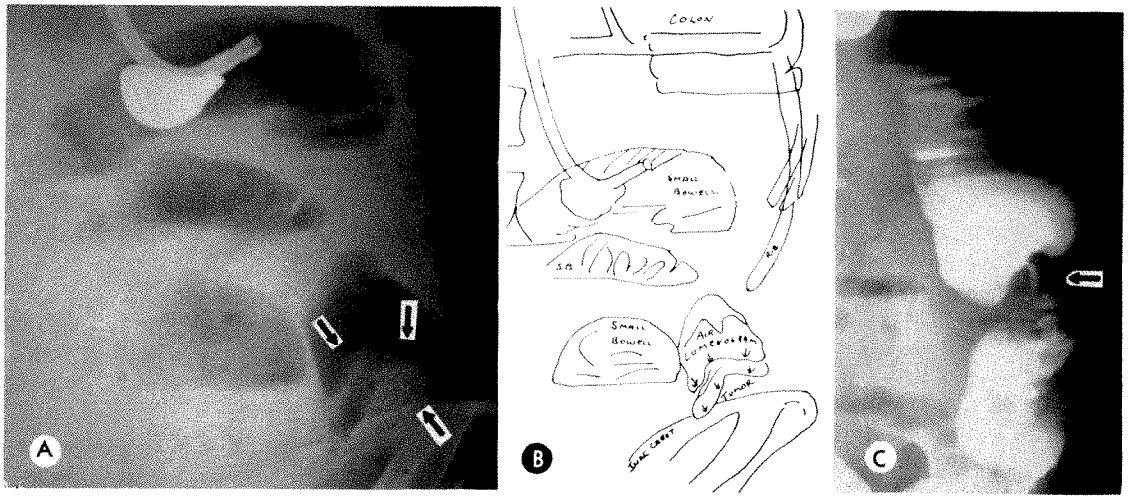


FIG. 9. Case IX. (*A*) Plain roentgenogram of the abdomen, April 26, 1959. The irregular polypoid or exophytic margins of the carcinoma of the descending colon project into the proximal air-filled segment of colon (arrows). Marked large and small bowel distention, with wide air-fluid levels in both, is seen. (*B*) Line drawing of *A*. The arrows indicate what is apparently the tumor mass of the descending colon projecting over the level of the left iliac crest. (*C*) Barium enema study, April 27, 1959. The stenotic lumen, caused by the annular carcinoma of the descending colon, is well demonstrated.

plasm of the stomach indicated an additional difficulty in visualizing the gastric "air lumenogram" due to the overlying lumbar spine which obscures the gas pattern on the frontal view.

The characteristic deformity in the air outline of the large bowel which is produced by a neoplasm may be of valuable ancillary assistance in the later evaluation of the barium enema studies.

CONCLUSION

Cases of various lesions of the gastrointestinal tract are presented in which the diagnosis was apparent on examination of the visceral gas patterns seen on plain abdominal roentgenograms, excretory urograms, roentgen studies of the lumbosacral spine, etc.) It is felt that a closer scrutiny of these roentgenograms will often lead to a presumptive diagnosis, which must then be confirmed by contrast studies. We do not believe that the "air lumenogram" can supersede the confirmatory gastrointestinal or barium enema studies, but it may, in some instances, serve as a valuable diagnostic aid.

S. Martin Bronson, M.D.
Department of Radiology
Carter County Memorial Hospital
Elizabethton, Tennessee

REFERENCES

1. CARMAN, R. D., and MILLER, A. The Roentgen Diagnosis of Diseases of the Alimentary Tract. W. B. Saunders Company, Philadelphia, 1917, p. 138.
2. FISCHER, M. S. Roentgen sign of gangrenous appendicitis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1959, *81*, 637–639.
3. FRIMANN-DAHL, J. Roentgen Examinations in Acute Abdominal Diseases. Charles C Thomas, Publisher, Springfield, 1951, p. 159.
4. KIRKLIN. Quoted by Ritvo, M., and Shauffer, I. A. In: Gastrointestinal X-Ray Diagnosis. Lea & Febiger, Philadelphia, 1952, pp. 218–219.

VOL. 86, NO. 1

THE EXAMINATION OF PATIENTS WITH SUSPECTED PERFORATED ULCER USING A WATER-SOLUBLE CONTRAST MEDIUM*

By GEORGE JACOBSON, M.D.,† CLARENCE J. BERNE, M.D.,‡ HARVEY I. MEYERS, M.D.,† *and* LEONARD ROSOFF, M.D.‡
LOS ANGELES, CALIFORNIA

UNTIL the present time, when examining patients suspected of having a perforated peptic ulcer, the function of the radiologist has been confined to the demonstration of pneumoperitoneum. Nothing beyond this has been necessary because the usual treatment of perforated peptic ulcer has been the immediate surgical closure of the perforation. There is, however, a growing concept that for selected patients nonoperative treatment may be a more suitable form of therapy.[1,2,7,8,10] Wangensteen[11] is credited with first suggesting, in 1935, that certain patients with perforated peptic ulcer might be treated only by aspiration of the stomach. In 1939, Mullen[4] cautiously applied this method to a small series of patients. Since then, larger series have been reported both in England and in this country. In 1957 Taylor,[9] one of the earliest and strongest advocates of this form of therapy, reported 256 patients with perforated ulcer of whom 21 were treated surgically and 235 by aspiration. The mortality of those treated by aspiration was about 2.5 per cent. Seeley and Campbell[6] reported 139 cases treated conservatively from 1945 to 1953. There were 7 deaths, giving an overall mortality of 5 per cent, or, by excluding 5 inoperable patients, a corrected mortality of 1.5 per cent. The same authors in reviewing the literature found 857 nonoperative cases, in which the mortality was 11.4 per cent with a corrected mortality, excluding 73 inoperable cases, of 4.5 per cent. The mortality in 1,622 operative cases reviewed by them was 8.7 per cent. Heslop *et al.*[3] reported a mortality rate of 7.7 per cent in a series of 104 nonoperative cases as compared with 8.6 per cent in 104 operative cases. In the 8 nonoperative deaths, 4 occurred in patients unsuited for surgery.

The nonoperative management of perforated peptic ulcer is predicated on the knowledge that many perforations have sealed when the patient is first seen. Sealing of the perforation by the direct apposition of adjacent structures is frequently observed even at early laparotomy. However, it is most difficult to differentiate clinically between those perforations which have recently sealed and those which continue to leak. In addition, errors in diagnosis constitute a serious problem since other conditions which simulate perforated peptic ulcer must be identified promptly so that proper treatment may be instituted.

It is evident that it is extremely advantageous to have a roentgenographic method that would demonstrate the presence or absence of leakage with a high degree of accuracy and which would also aid in the identification of other conditions simulating perforated peptic ulcer. The radiologist has been understandably reluctant to administer barium sulfate for fear that it might spill into the peritoneal cavity. Although water-soluble contrast media have been used to examine the gastrointestinal tract with increasing frequency, we have encountered only one reference to their use in conjunction with perforated ulcer. Rea[5] reported in 1952 that he had administered 10 cc. of diodrast to 5 patients and ob-

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.
The contrast medium used in this study was supplied by Winthrop Laboratories, New York, New York.
From the Departments of Radiology† and Surgery,‡ University of Southern California School of Medicine, and the Los Angeles County Hospital.

tained supine and upright roentgenograms of the abdomen. Diodrast was seen in the peritoneal cavity in 1 case.

Early in 1958, at the suggestion of one of the authors (C.J.B.), a study was begun to evaluate the incidence of spontaneous sealing of perforated peptic ulcers. A water-soluble contrast substance, hypaque, was instilled through a nasogastric tube as part of this study to determine roentgenologically whether or not sealing of the ulcer had occurred. It is the purpose of this paper to describe the technique of the examination and the roentgenographic findings in 120 patients. The clinical results of this study will be reported separately.

TECHNIQUE OF EXAMINATION

For a number of years in our department, the roentgenologic examination of each patient suspected of having a perforated intra-abdominal viscus has included an upright posteroanterior roentgenogram of the chest and two roentgenograms of the abdomen, consisting of an anteroposterior supine view and lateral decubitus view with the right side up. We believe a chest roentgenogram should be part of the examination of all patients with acute abdominal symptoms, not only to identify free subdiaphragmatic gas but also pulmonary or cardiac lesions which might be responsible for the patient's symptoms or complicate the situation.

Our first examination of the stomach and duodenum using a water-soluble contrast medium was fairly simple. After the preliminary roentgenograms had been obtained, 60 cc. of 50 per cent intravenous hypaque was instilled into the stomach through a Levin tube. Two anteroposterior roentgenograms of the abdomen were then made, one supine and the other with the patient in the semi-erect position. It soon became apparent that with this technique, leakage of fairly large amounts of hypaque into the peritoneal cavity could be detected easily. In one or two instances, the site of perforation was seen, but, in general, the stomach and duodenum were not well visualized. It was then decided to extend the examination, fluoroscoping each patient and obtaining spot roentgenograms if possible. This proved to be easily accomplishable and the examination soon developed into a modified but thorough upper gastrointestinal examination.

As noted above, our first examinations were performed with the intravenous preparation of hypaque. While this was quite satisfactory as a contrast medium, it was rather expensive since it was necessary to use at least two, and sometimes three vials. We were able to obtain powdered hypaque in bulk form and found, after some experimentation, that 50 gm. dissolved in 75 cc. of sterile water made a solution of ideal concentration. This volume has proved sufficient to perform most examinations. Several samples of powdered hypaque were examined bacteriologically. None contained any pathogenic bacteria. Ten drops of a wetting agent (tween 80) have been added to aid in the coating of the mucosa. All of the water-soluble contrast media are very bitter and cannot be administered orally unless a flavoring agent is added. We have not attempted to do this for such a substance might prove irritating to the peritoneal cavity. We did not believe it was necessary inasmuch as the stomach is emptied by nasogastric suction and the tube left in place in each patient suspected of having an acute abdominal lesion before the patient is brought to the radiology department.

The examination is now performed as follows: After the preliminary roentgenograms for pneumoperitoneum have been obtained, the patient who previously has been given a narcotic is placed on a fluoroscopic table and the solution of hypaque is given through the Levin tube. The patient is fluoroscoped carefully. If there is an obvious massive leak of contrast medium into the peritoneal cavity, spot roentgenograms are made rapidly to record the site of leakage and to determine, if possible, the nature of the lesion which has produced the perforation. Supine and semi-erect 14×17

inch roentgenograms are then made. If there is no massive leakage, the stomach and duodenum are examined in much the same manner as for a conventional upper gastrointestinal study. Multiple spot and the usual over-the-table Bucky roentgenograms are made, followed by supine and semi-erect 14×17 inch roentgenograms. The examination may be extended readily to include the small intestine when, for example, it becomes evident that the patient's symptoms are due to intestinal obstruction, etc.

Almost all the patients we have examined have been able to cooperate fully, even though many have been aged and critically ill. In only a few instances have we had to modify the study because of the patient's condition.

RESULTS

Patients suspected of having a perforated peptic ulcer, who are examined roentgenologically, while acutely ill, fall into the following groups: (1) gastroduodenal ulcer with or without leakage of contrast medium from the ulcer site, with pneumoperitoneum; (2) gastroduodenal ulcer with or without leakage of contrast medium from the ulcer site without pneumoperitoneum; (3) other conditions with pneumoperitoneum simulating perforated peptic ulcer, with or without gastroduodenal ulcer; and (4) other conditions without pneumoperitoneum simulating perforated peptic ulcer, with or without gastroduodenal ulcer.

For purposes of simplification, the term peptic ulcer has been used to include not only duodenal and benign gastric ulcer, but also malignant gastric ulcer. It was determined (Table 1) that 98 (82 per cent) of the 120 patients examined had a peptic ulcer which was responsible for their symptoms and 82 of these patients had a definite perforation in that there was pneumoperitoneum and/or leakage of contrast material from an ulcer. Excluding the 16 cases with neither pneumoperitoneum nor leakage, 93 per cent of the patients with perforated peptic ulcer had pneumoperitoneum and 50 per cent of the ulcers were leaking when examined. Of particular importance are the 6 patients, 7 per cent of those with proved perforated ulcer, who had leakage of contrast material from their ulcer in the absence of pneumoperitoneum.

Twenty-two (18 per cent) of the 120 patients had some other conditions which clinically simulated perforated peptic ulcer. Of these 9 (41 per cent) had pneumoperitoneum and 13 (59 per cent) did not. The various diagnoses are listed in Table 1. It is of interest that 6 of the 9 patients with pneumoperitoneum were females while all 13 patients without pneumoperitoneum were males. This is perhaps due to the fact

TABLE I

SUSPECTED PERFORATED PEPTIC ULCER
120 PATIENTS
Males 99 (83%) Females 21 (17%)

Final Diagnosis—Peptic Ulcer—98 (82%) Males 83 (85%) Females 15 (15%)			
Pneumoperitoneum—no leakage			41 (42%)
Pneumoperitoneum—leakage			35 (36%)
No pneumoperitoneum—no leakage			16 (16%)
No pneumoperitoneum—leakage			6 (6%)
Final Diagnosis—No Peptic Ulcer—22 (18%)			
	Males	Females	
Pneumoperitoneum			
Diagnosis undetermined	2	1	
Perforated diverticulum of colon	1	4	
Perforated carcinoma of colon	—	1	
	3	6	9 (41%)
No Pneumoperitoneum			
Pancreatitis	3	—	
Cholecystitis	2	—	
Undetermined	2	—	
Perforated diverticulum of colon	1	—	
Traumatic rupture of jejunum	1	—	
Carcinoma of duodenum	1	—	
Intestinal obstruction	1	—	
Alcoholic gastritis	1	—	
Perforated appendix	1	—	
	13	—	13 (59%)

TABLE II

LEAKAGE OF CONTRAST MEDIUM IN 82 PATIENTS WITH PROVED PERFORATION

		No Leakage	Leakage
Duodenal Ulcer—65 (Males 56, Females 9)			
Pneumoperitoneum	61 (94%)	33	28
No Pneumoperitoneum	4 (6%)	—	4
Total	65 (100%)	33 (50%)	32 (50%)
Benign Gastric Ulcer—14 (Males 9, Females 5)			
Pneumoperitoneum	13	8	5
No Pneumoperitoneum	1	—	1
Malignant Gastric Ulcer—3 (Males 3,* Females 0)			
Pneumoperitoneum	2	—	2
No Pneumoperitoneum	1	—	1

* Leakage in all 3 cases confined to area of perforation.

that perforation of a colonic diverticulum, the second most common cause of pneumoperitoneum, in our experience is more frequent in females than males.

Tables II and III summarize the occurrence and extent of leakage of contrast material from the 3 categories of gastroduodenal ulcer and the relationship of pneumoperitoneum to leakage. Leakage was found to occur in 4 distinct patterns: (1) generalized, spreading throughout all or most of the peritoneal cavity; (2) confined to the right upper quadrant or the liver edge; (3) confined to the left upper quadrant; and (4) confined to the immediate area of perforation.

Duodenal Ulcer. There were 65 patients with proved perforated duodenal ulcer, 61 (94 per cent) of whom had pneumoperitoneum. Leakage of contrast material occurred in 32 of these patients or 50 per cent of those with subsequently proved perforation. In almost one-half of these 32 patients, the leage was generalized (Fig. 1, *A* and *B*). In 10 patients it was confined to the right upper quadrant or liver edge (Fig. 2, *A* and *B*); and, in 7, the leakage was localized to the area of perforation (Fig. 3, *A* and *B*). Leakage occurred in 4 cases in which there was no demonstrable pneumoperitoneum, and in one of these, the leakage was generalized (Fig. 4, *A*, *B* and *C*). There were no cases in which leakage from the perforated duodenal ulcer was confined to the left upper quadrant of the abdomen.

TABLE III

EXTENT OF LEAKAGE OF CONTRAST MATERIAL

	Duodenal Ulcer		Benign Gastric Ulcer		Malignant Gastric Ulcer	
	Pneumo-peritoneum	No Pneumo-peritoneum	Pneumo-peritoneum	No Pneumo-peritoneum	Pneumo-peritoneum	No Pneumo-peritoneum
Generalized	14	1	3	—	—	—
Right Upper Quadrant	8	2	—	—	—	—
Left Upper Quadrant	—	—	2	1	—	—
Area of Perforation	6	1	—	—	2	1
Total	28	4	5	1	2	1

Benign Gastric Ulcer. Of the 14 cases of benign gastric ulcer, all of which were perforated, 13 had pneumoperitoneum. Leakage occurred in 6 cases; it was generalized in 3 instances and in the remainder, one of which did not have pneumoperitoneum, it was confined to the left upper quadrant (Fig. 5, *A*, *B* and *C*). There were no cases in which the leakage was localized to the right upper quadrant or in the area of perforation.

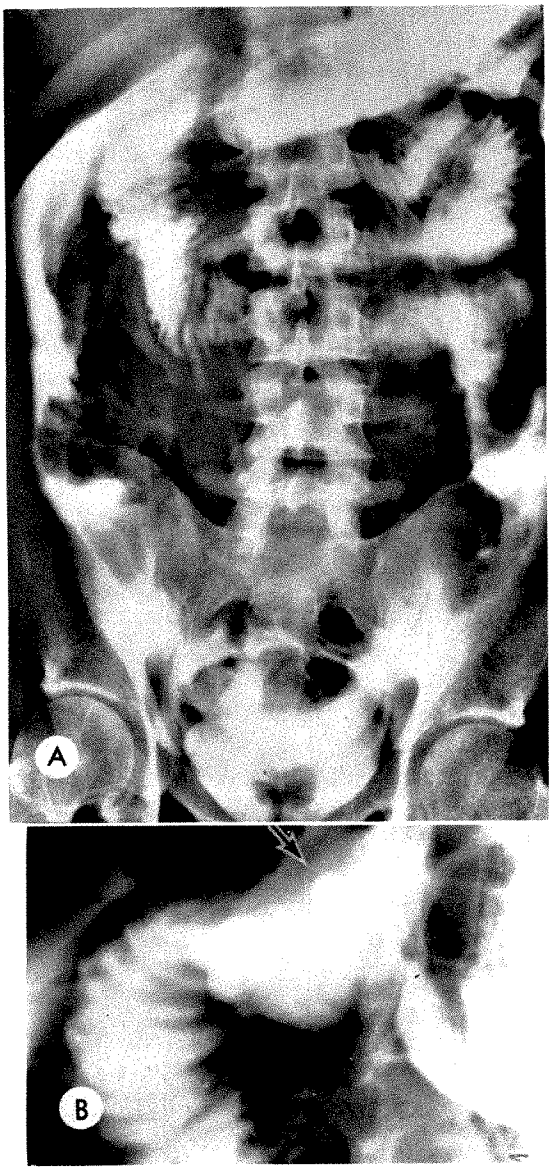


FIG. 1. Twenty year old male with acute symptoms of 2 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer with massive leakage and urinary tract excretion and (*B*) an ulcer crater on the spot roentgenogram (arrow). Final diagnosis: duodenal ulcer with 1.0 cm. open perforation (surgery).

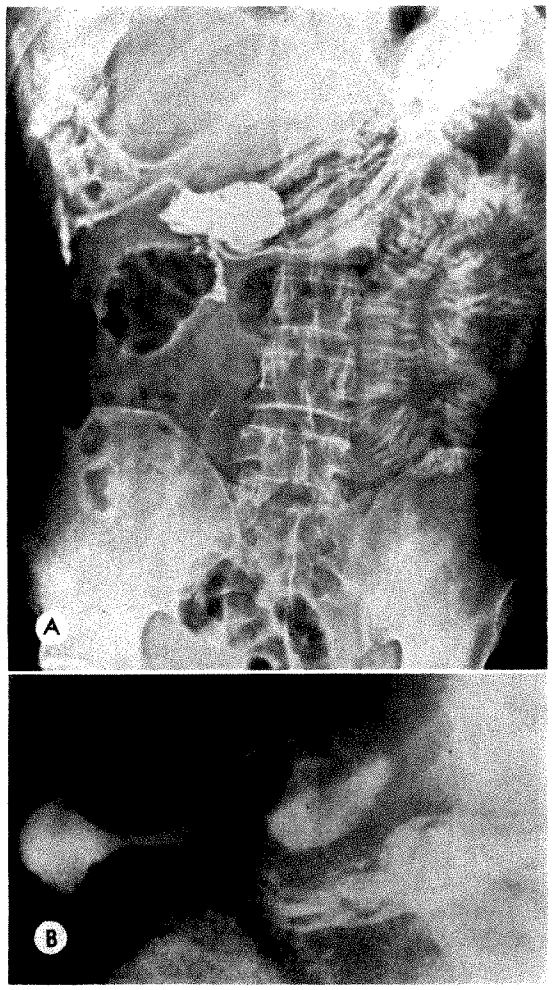


FIG. 2. Fifty-seven year old male with acute symptoms of 12 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer tract along the lower edge of the liver, leakage confined to the right upper quadrant and (*B*) two days later, persistence of leakage. Final diagnosis: duodenal ulcer with 8 mm. open perforation (surgery).

Age and Sex. No relationship could be demonstrated between the age or sex of the patient and leakage of contrast material. The age and sex of those in whom leakage was demonstrated was not signifi-

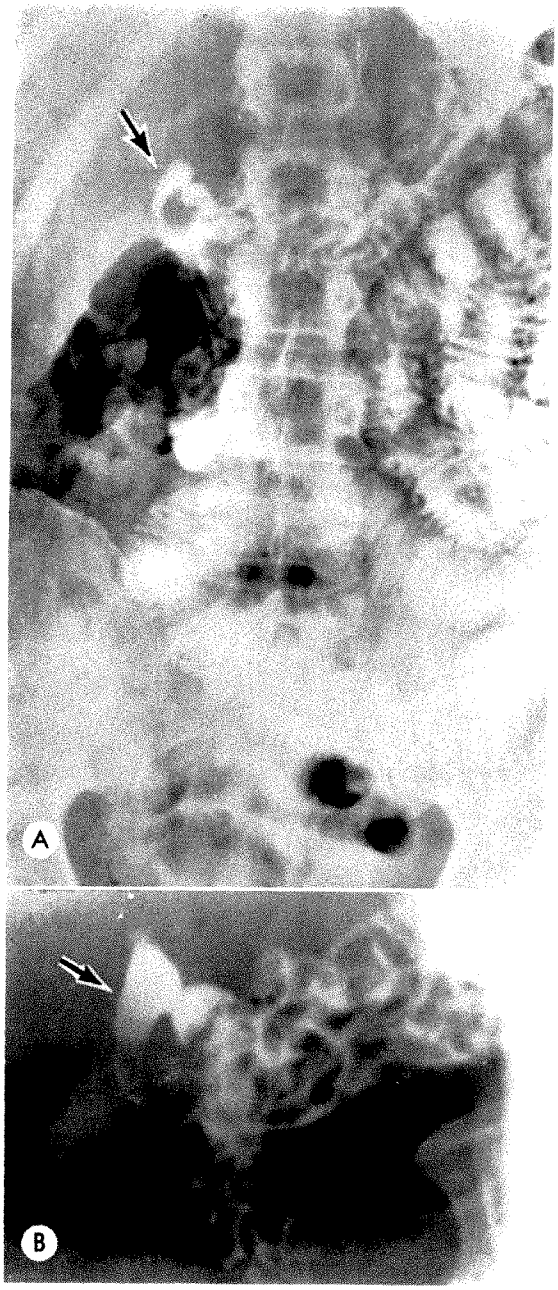


FIG. 3. Forty year old male with acute symptoms of 6 hours' duration and previous symptoms of 10 months' duration. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer crater with leakage confined to the area of perforation (arrow) and (*B*) a collection of hypaque (arrow) adjacent to the ulcer crater on the spot roentgenogram. Final diagnosis: duodenal ulcer with localized perforation (elective surgery).

cantly different from that of the entire group with perforated ulcer.

Duration of Symptoms. It was thought that the duration of ulcer symptoms or symptoms of acute perforation might have some bearing on whether the perforation would have sealed spontaneously by the time the examination was performed, *i.e.*, an acute ulcer might be expected to seal spontaneously more readily than a chronic ulcer. As shown in Table IV, there were 10 patients with perforated duodenal ulcer who had no previous ulcer symptoms; in 6 of these leakage was noted. On the other hand, no leakage could be demonstrated in 21 of the 41 patients with perforated duodenal ulcer who had ulcer symptoms for more than six months.

The length of time between the onset of acute symptoms of perforation and the roentgen examination of the stomach and duodenum is shown in Table V. Slightly more than one-half the patients with perforated duodenal ulcer examined within six hours and a somewhat smaller proportion of those examined from six to twelve hours after the onset of symptoms had leakage when examined. However, there was no significant difference in the percentage of unsealed ulcers found in patients examined either twelve to twenty-four hours or even more than forty-eight hours after the onset of symptoms.

The much smaller group of benign gastric ulcers does not appear to differ significantly from the perforated duodenal ulcer group in the relationship of duration of ulcer symptoms and symptoms of acute perforation to spontaneous sealing.

Malignant Gastric Ulcer. In distinct contrast were the 3 cases of malignant gastric ulcer. Leakage was present in all 3 of these cases, one of which did not have pneumoperitoneum, and in each the leakage was confined to the area of perforation (Fig. 6, *A* and *B*).

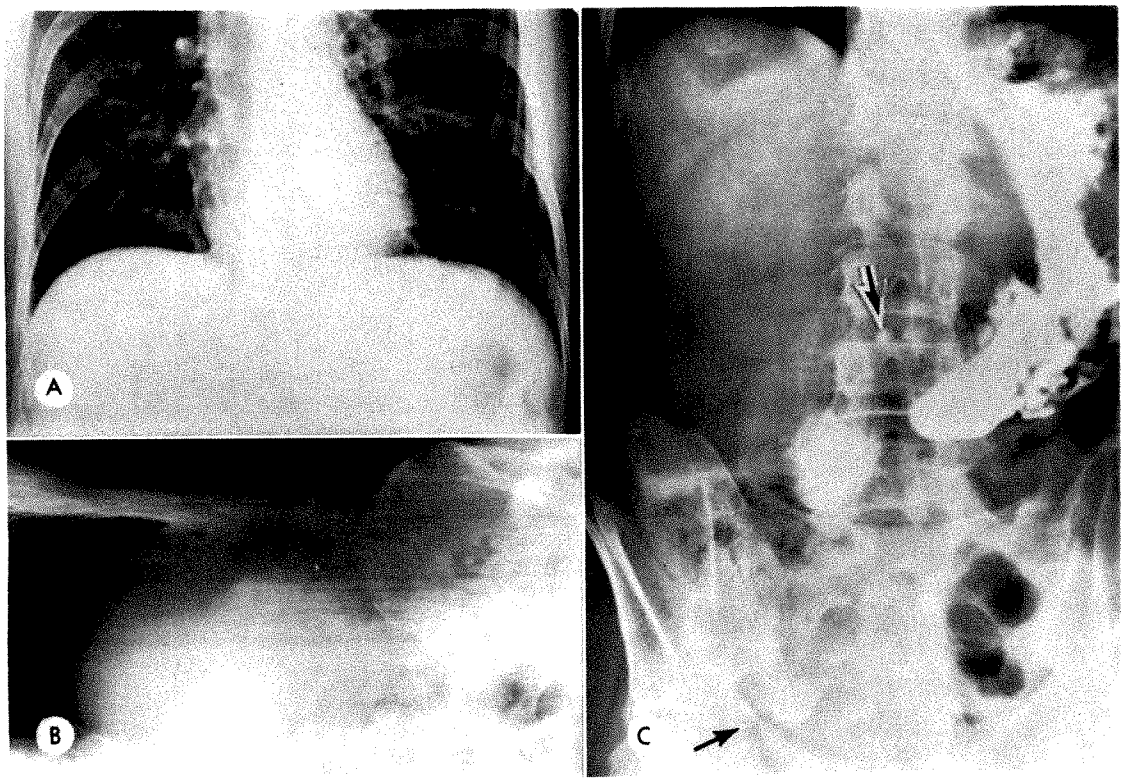


FIG. 4. Fifty-four year old male with acute symptoms of 2 hours' duration and previous symptoms of 7 days' duration. Upright chest and lateral decubitus roentgenogram showed (*A* and *B*) no evidence of pneumoperitoneum. Hypaque study revealed (*C*) that the contrast material coated the bowel in the right lower quadrant and pelvis (lower arrow). Upper arrow points to the ulcer crater. Final diagnosis: duodenal ulcer with 0.5 cm. open perforation (surgery).

TABLE IV

RELATION OF ULCER SYMPTOMS TO LEAKAGE OF CONTRAST MATERIAL

Duration of Symptoms (mo.)	No Leakage	Generalized Leakage	Leakage in Right Upper Quadrant	Leakage in Left Upper Quadrant	Leakage at Area of Perforation
			Duodenal Ulcer		
0	4	2	4	—	—
0–3	7	3	1	—	2
3–6	—	—	—	—	—
6	21	10	5	—	5
No History	1	—	—	—	—
			Benign Gastric Ulcer		
0	—	1	—	—	—
0–3	4	2	—	—	—
3–6	—	—	—	—	—
6	4	—	—	2	—
No History	—	—	—	1	—

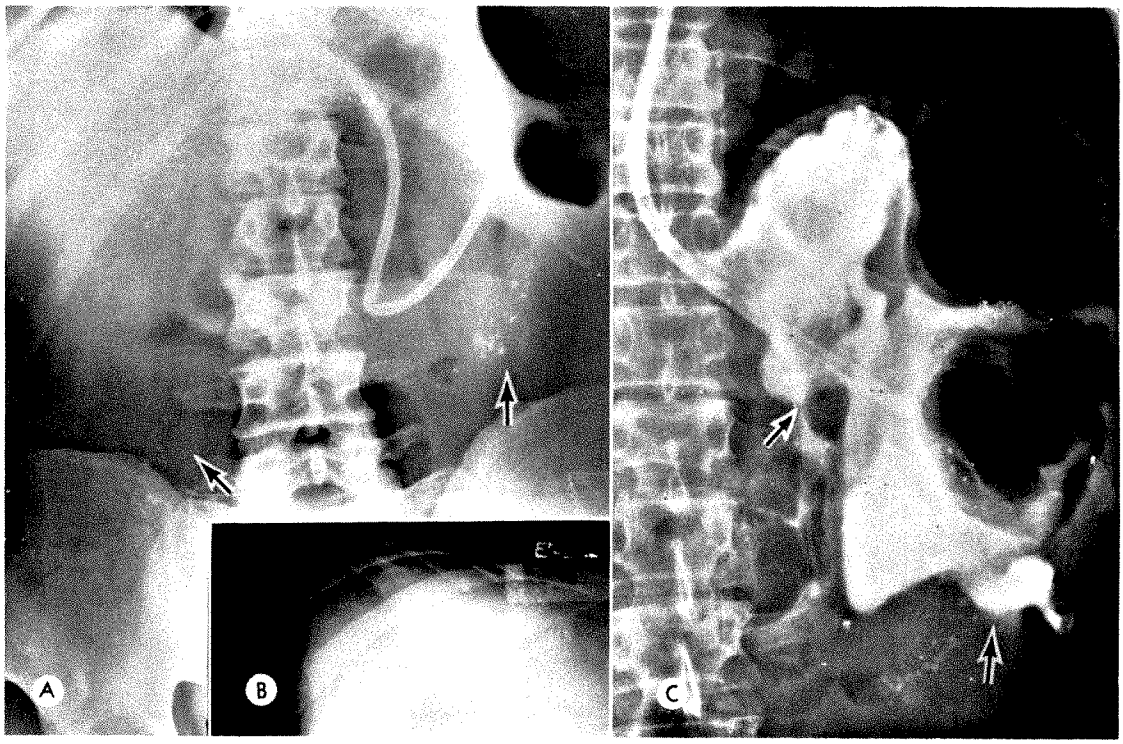


FIG. 5. Forty-seven year old male with no available history. (*A* and *B*) Loculated gas in the left upper quadrant was thought at first to be in the splenic flexure of the colon. Arrows point to pancreatic calculi. Differential diagnosis: pancreatitis versus perforated ulcer. (*C*) Hypaque study shows large gastric ulcer (upper arrow) with massive leakage (lower arrow) confined to left upper quadrant. Final diagnosis: perforated 2.5 cm. lesser curvature gastric ulcer.

TABLE V

RELATION OF DURATION OF SYMPTOMS OF PERFORATION TO LEAKAGE OF CONTRAST MATERIAL

Duration of Symptoms (hr.)	No Leakage	Generalized Leakage	Leakage in Right Upper Quadrant	Leakage at Area of Perforation
		Duodenal Ulcer		
0–6	14	9	4	4
6–12	11	1	2	2
12–24	6	3	1	1
24–48	0	1	1	0
48	2	1	2	0
		Benign Gastric Ulcer		
0–6	1	1	—	—
6–12	2	—	1	—
12–24	2	2	—	—
24–48	—	—	1	—
48	3	—	—	—
No History	—	—	1	—

Urinary Tract Excretion. Excretion of opaque material through the urinary tract was observed in several cases in which there was generalized leakage of contrast material into the peritoneal cavity. Hypaque is not appreciably absorbed from the gastrointestinal tract. The absorption presumably occurred through the peritoneum.

Diagnoses. All examinations have been included except several performed early in the development of the procedure which were technically unsatisfactory. Since modifying our technique to more closely resemble a conventional upper gastrointestinal examination and overcoming our reluctance to examine this type of patient, the studies have improved rapidly in quality and at present are rather uniformly good. All the examinations have been performed by the resident staff.

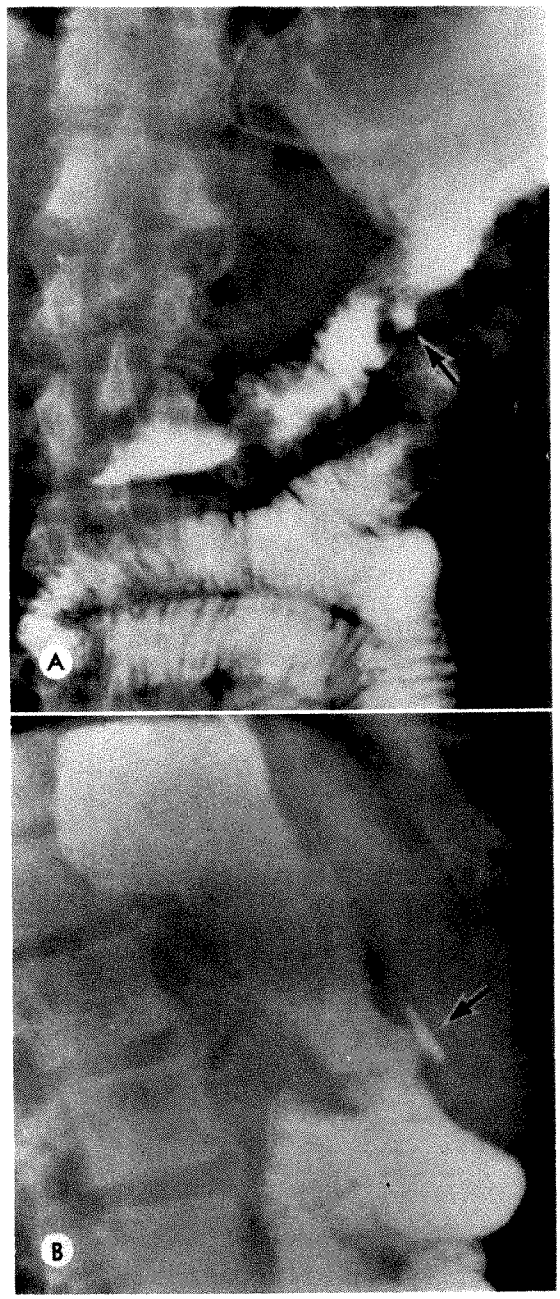


Fig. 6. Eighty year old male with acute symptoms of 1 day's duration and previous symptoms of 9 months' duration. Pneumoperitoneum was demonstrated. Hypaque studies showed (*A*) a greater curvature ulcer with surrounding filling defect (arrow) and (*B*) a small anterior localized perforation (arrow). Final diagnosis: malignant gastric ulcer with localized perforation.

»»→

Table VI indicates the manner of arriving at a final diagnosis. Each case is listed only once under the most reliable means of confirmation. All but 15 cases were confirmed by surgery (immediate or elective), follow-up gastrointestinal examinations, or autopsy. In these 15 patients, the final diagnoses were based on the hypaque studies and other roentgenologic examinations and quite unequivocal clinical findings.

There were 4 errors in roentgenologic diagnosis (Table VII). While the absence of leakage was successfully demonstrated in these cases, they were considered errors to the extent that the duodenal and gastric ulcers were not demonstrated by the hypaque studies. A sealed perforation was confirmed by surgery in 3 cases (Fig. 7, *A* and *B*) and in the fourth, a follow-up gastrointestinal examination demonstrated a duodenal ulcer.

COMMENT

The technique described in this paper of using a water-soluble contrast medium to examine the stomach and duodenum of a patient suspected of having a perforated peptic ulcer was developed in conjunction with the nonoperative management of perforated peptic ulcer and was prompted by the difficulty of recognizing clinically those cases in which the perforation continues to leak. The authors of previously published reports dealing with the conservative management of perforated peptic ulcer have assumed that all patients included had a defi-

Table VI

CONFIRMATION OF DIAGNOSIS

	Surgical Findings*	Follow-up Gastrointestinal Studies	Clinical Findings	Autopsy Findings
Ulcer	73	12	12	1
No Ulcer	12	5	3	2

* Includes cases confirmed by elective surgery.

TABLE VII

ERRORS IN DIAGNOSIS (4 CASES)

Sex	Age	Duration of Acute Symptoms (hr.)	Duration of Previous Symptoms	Pneumo-peritoneum	Hypaque Study	Final Diagnosis
M	31	14	0	Yes	Normal, no leakage	Giant bleeding duodenal ulcer, sealed perforation (surgery)
M	50	10	0	No	Normal, no leakage	Duodenal ulcer (follow-up gastrointestinal study)
M	49	10	5 yr.	Yes	Normal, no leakage	Gastric ulcer, sealed perforation (follow-up gastrointestinal study and surgery)
M	30	2	2 wk.	Yes	Normal, no leakage	Gastric ulcer, sealed perforation (surgery)

nite perforation. However, there are those patients who develop acute symptoms simulating perforation strongly enough to be diagnosed as such without perforation actually being present. There were 16 patients (15 with duodenal ulcer and 1 with malignant gastric ulcer) in the present study who had neither pneumoperitoneum nor leakage of contrast material. It is quite likely that these cases represent penetrating rather than perforated ulcers.

The danger of misdiagnosis has been of considerable concern to all. In the present series, 18 per cent of the patients proved to have some condition not related to peptic ulcer. Some of these patients required prompt surgical intervention. It is obvious that the criteria used for selecting patients for the examination will determine the proportion of nonulcer patients in any given group such as ours.

Leakage was demonstrated in almost 50 per cent of the 65 cases of perforated duodenal ulcer, in a slightly smaller number, 43 per cent (6 of the 14 cases) of perforated benign gastric ulcer, and in all 3 cases of perforated malignant gastric ulcer. All patients were examined as soon after admission to the hospital as feasible and all those with generalized intraperitoneal leakage were operated upon promptly. However, for one reason or another, surgery was delayed in several patients with localized leakage. Two of these patients were reexamined. One had a perforated malignant gastric ulcer with leakage confined to the area of perforation. Two days later, the ulcer was shown to have sealed (Fig. 8, *A* and *B*). The other patient had a perforated duodenal ulcer with the leakage confined to the right upper quadrant and a well-defined tract along the liver edge. Reexamination three days later showed continued leakage from the ulcer and the patient was operated upon (Fig. 2, *A* and *B*). It should be stressed that of those examined more than twelve hours after the onset of symptoms, about one-half were found to have leakage.

It has been suggested by Taylor[9] and Seeley and Campbell,[6] among others, that acute ulcers, particularly in young individuals, can be expected to seal spontaneously and that perforated chronic ulcers often do not. Our results do not bear this out. The proportion of ulcers leaking at the time of examination was the same for patients with a history of ulcer symptoms of long duration as for those with a history of short duration. The incidence of leakage

did not vary significantly with the time which had elapsed between the onset of symptoms of perforation and the performance of the roentgen examination. This suggests that, perhaps, those perforations which are going to seal spontaneously do so very soon after the perforation has occurred.

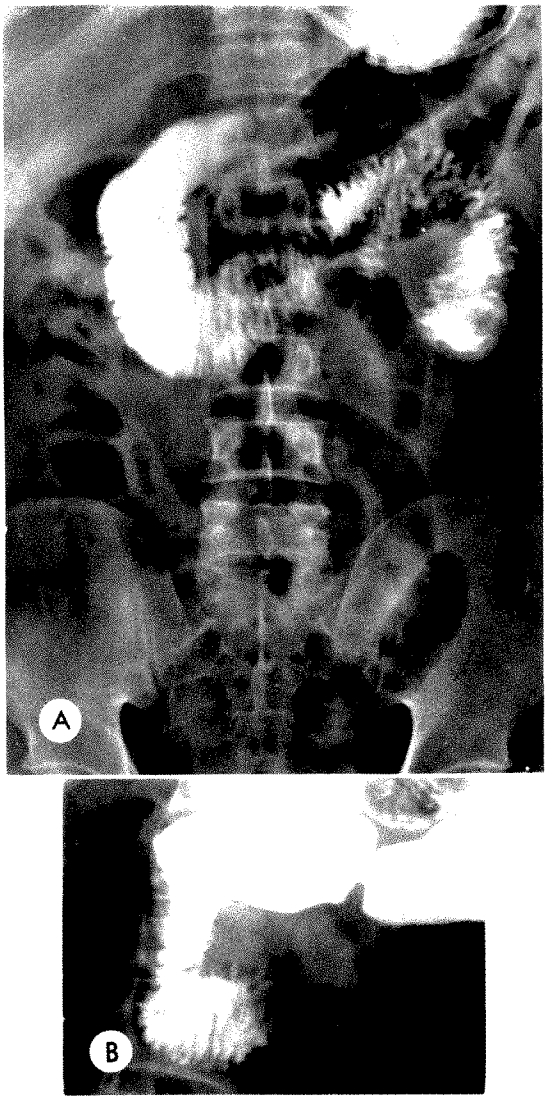


Fig. 7. Thirty-one year old male with acute symptoms of 14 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) no leakage and (*B*) a normal duodenal bulb on spot roentgenogram. Final diagnosis: giant bleeding duodenal ulcer (delayed surgery).

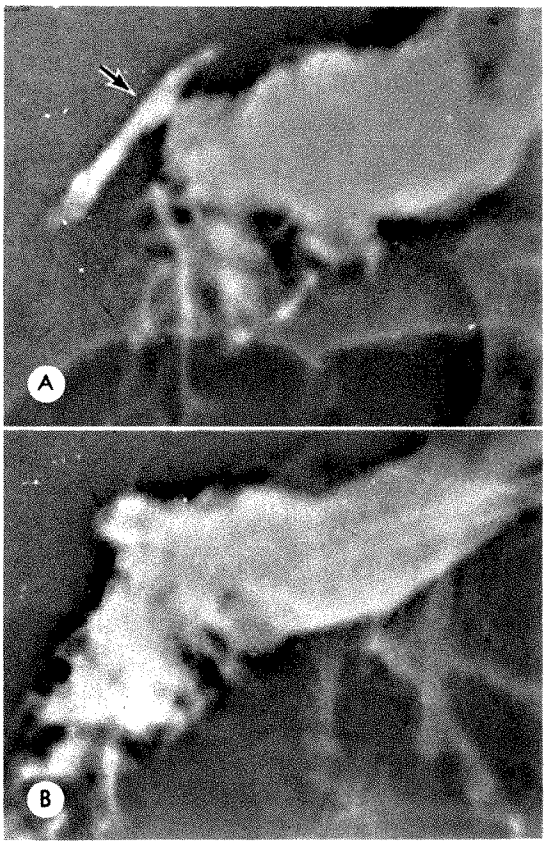


Fig. 8. Seventy-one year old male with acute symptoms of 36 hours' duration and previous symptoms of 1 year's duration. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a large irregular gastric ulcer (arrow) with leakage confined to the area of perforation and (*B*) a sealed perforation on a repeat examination 2 days later. Final diagnosis: malignant gastric ulcer with sealed perforation (elective surgery).

Perforation of peptic ulcer in the absence of demonstrable pneumoperitoneum is particularly difficult to diagnose clinically, and even more difficult to prove. Possibly some of the 16 patients with neither pneumoperitoneum nor leakage had "forme fruste" perforations, though to be conservative we have assumed that they were not perforated. However, there were 6 patients without pneumoperitoneum with leakage of the ulcers at the time of examination; 4 from duodenal ulcers and 1 each from benign and malignant gastric ulcers. What is of considerable importance is that

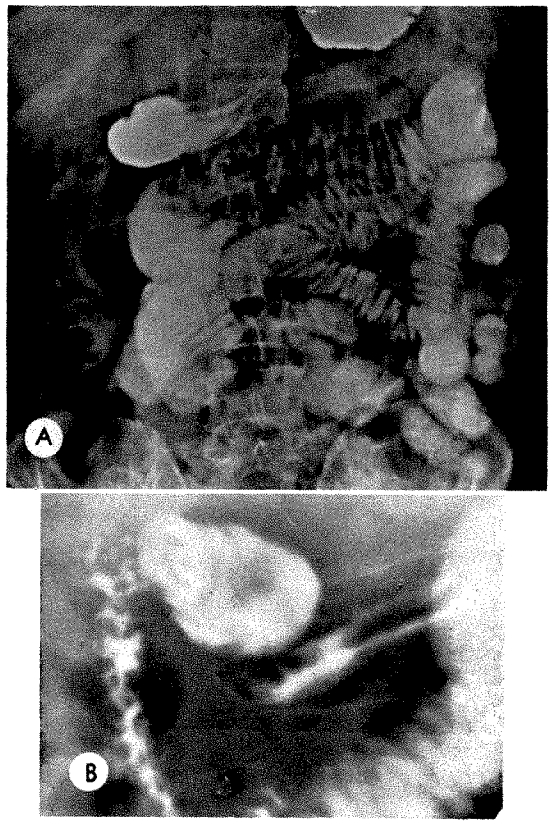


FIG. 9. Forty-eight year old male with acute symptoms of 24 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a normal stomach and duodenum and no leakage and (*B*) a normal duodenal bulb on spot roentgenogram. Final diagnosis: perforated diverticulum of colon (surgery).

in one of these patients with an unsealed perforated duodenal ulcer the leakage was generalized.

The distribution and pathways of leakage of gastrointestinal contents from perforated peptic ulcer have long been known from surgical and postmortem observations. The procedure described in this paper has afforded the first opportunity to demonstrate these pathways visually. At the time of examination, generalized leakage throughout the peritoneal cavity was present in almost one-half of the patients with leaking duodenal and gastric ulcers. Leakage from perforated duodenal ulcers is frequently confined to either the right upper quadrant, the liver edge, or the area of perforation and from perforated benign gastric ulcer to the left upper quadrant. No localization to the right upper quadrant or area of perforation was observed in the small series of perforated benign gastric ulcers. It is also of interest that the leakage in all 3 cases of perforated malignant gastric ulcer was confined to the area of perforation.

As already noted, the examination has not been technically difficult in spite of the fact that many of the patients are of the older age group and seriously ill. When performed carefully and without delay, the procedure is a safe and accurate means of demonstrating continued leakage from a perforated peptic ulcer, and the nature and location of the lesion. The demonstration of a normal stomach and duodenum aids in the prompt identification of those patients whose clinical picture simulates perforated peptic ulcer but who have some other condition (Fig. 9, *A* and *B*). It should be emphasized that, as in the 4 cases listed in Table V, there are those instances in which the ulcer crater is not demonstrated. However, there is no assurance that one could have shown these ulcers with a barium study. As a matter of fact, it is our impression that it is easier to demonstrate an ulcer crater, particularly of the duodenal bulb, with a water-soluble contrast medium than with barium.

SUMMARY

The roentgenographic determination of spontaneous sealing of perforated peptic ulcer has an important place in the nonoperative managment of this condition. A modified but thorough examination of the upper gastrointestinal tract using a water-soluble contrast medium was performed on 120 patients with suspected perforation.

Approximately 50 per cent of the proved perforated duodenal and benign gastric ulcers and all of the proved perforated malignant gastric ulcers were leaking at the time of examination. The leakage was generalized throughout the peritoneal cavity in almost one-half of these patients.

Leakage of contrast material was demonstrated in 6 cases (7 per cent) of perforated ulcer (4 duodenal, 1 benign gastric, 1 malignant gastric) in which there was no pneumoperitoneum.

The proportion of perforated ulcers which were sealed was not affected by age, sex, duration of symptoms of perforation, or chronicity of the ulcer.

By demonstrating a normal stomach and duodenum, the examination aided in the prompt identification of patients who had other conditions simulating perforated peptic ulcer.

The technique of examination is described. The examination is not difficult to perform. No patients have been harmed by the procedure.

George Jacobson, M.D.
1200 North State Street
Los Angeles 33, California

REFERENCES

1. Berne, C. J., and Mikkelsen, W. P. Management of perforated peptic ulcer. *Surgery*, 1958, *44*, 591–603.
2. Editorial: Treatment of perforated peptic ulcer. *Lancet*, 1951, *260*, 36–38.
3. Heslop, T. S., Bullough, A. S., and Brun, C. Treatment of perforated peptic ulcer; comparison of parallel unselected series. *Brit. J. Surg.*, 1952, *40*, 52–58.
4. Mullen, B. P. Quoted by Rea, C. E.[5] and Taylor, H.[9]
5. Rea, C. E. Conservative versus operative treatment of perforated peptic ulcer. *Surgery*, 1952, *32*, 654–657.
6. Seeley, S. F., and Campbell, D. Nonoperative treatment of perforated peptic ulcer; further report. *Internat. Abstr. Surg.*, 1956, *102*, 435–446.
7. Stead, J. R., St. G. Conservative treatment of perforated peptic ulcer. *Lancet*, 1951, *260*, 12–17.
8. Taylor, H. Aspiration treatment of perforated ulcers; further report. *Lancet*, 1951, *260*, 7–12.
9. Taylor, H. Non-surgical treatment of perforated peptic ulcer. *Gastroenterology*, 1957, *33*, 353–368.
10. Vaughan, R. H., and Warren, R. Perforated peptic ulcer; study of indications for non-surgical treatment in selected cases. *New England J. Med.*, 1951, *244*, 898–902.
11. Wangensteen, O. H. Non-operative treatment of localized performations of duodenum. *Minnesota Med.*, 1935, *18*, 477–480.

SIMPLE BENIGN PREPYLORIC ULCER*

THE POSSIBILITY OF AN UNEQUIVOCAL ROENTGEN DIAGNOSIS

By BERNARD S. WOLF, M.D., *and* DAVID BRYK, M.D.
NEW YORK, NEW YORK

THE report of a benign prepyloric gastric ulcer on roentgen examination is accepted by the referring physician or consulting surgeon with considerable reservation because of skepticism that the radiologist can differentiate between benign and malignant ulceration in this part of the stomach. This belief apparently took firm hold in 1933 as a result of a report by Hampton[4] that all prepyloric ulcers observed at the Massachusetts General Hospital in a three year period were carcinomatous. This author therefore considered that the location of the ulceration was of overwhelming diagnostic importance, to the exclusion of all other features. In contrast, Singleton[13] in 1936 reported that in any given case of an ulcer within one inch of the pyloric sphincter, the probability was about four to one that it was benign. Doub[3] in 1940 studied 35 cases of ulceration of the pyloric and prepyloric areas believed to be benign or indeterminate on roentgen examination. These cases either failed to respond to medical management or were so extensive that a suspicion of carcinoma had to be entertained. Twenty-four turned out to be benign on pathologic study. Kirklin and MacCarthy[6] in 1942 reviewed all cases of ulceration of the prepyloric segment of the stomach operated upon at the Mayo Clinic from 1937 to 1941. Of a total of 132 cases, 63 were benign ulcers, 8 malignant ulcers and 61 ulcerated carcinomas. In 47 instances in which a niche was seen without evidence of tumefaction or other indication of malignancy, 24 cases were benign, 8 malignant and 5 were duodenal ulcers. These authors concluded that prepyloric ulcers were not more likely to be carcinomatous than ulcers situated elsewhere in the stomach. Collins[2] in 1952 analyzed 139 prepyloric lesions confirmed by surgery or follow-up. Of 54 cases said to be benign on roentgen examination, only 8 were proved to be malignant.

It is evident from the above brief and incomplete summary of the literature that the original report of Hampton has been contradicted many times. It is nevertheless surprisingly difficult to eradicate the impression originally made. It is not entirely clear why the report of Hampton is at such great variance with subsequent careful studies. One of the reasons may be that, as Hampton emphasized, he included only chronic cases of prepyloric ulceration that were associated with distinct induration. Since the emphasis in this article was on location of the lesion, the roentgen features were not described. In many of the reports which attempt to assess the accuracy of roentgen diagnosis in this area, evaluation is difficult because the effort made to demonstrate specific features and the degree of success in delineating them have necessarily varied a great deal. Moreover, from the roentgen point of view, the possibility of a correct differential diagnosis depends a great deal on the type of benign ulcer, *i.e.*, whether it is small and nonpenetrating or whether it is large and calloused. A penetrating ulcer completely destroys the muscularis propria layer and produces a large amount of granulation tissue and fibrous reaction which simulates roentgenologically, and often at exploration, a mass lesion. In contrast, a "simple" ulcer, that is, an ulceration which has not extended through the muscularis propria rarely presents as a pseudotumor unless it is relatively acute and associated with marked surrounding edema. In assessing the accuracy of roentgen diagnosis, it is essential to distinguish

* From the Department of Radiology of The Mount Sinai Hospital, New York, New York.

between these two types of benign ulceration. In the large majority of instances, it should be possible to recognize a simple ulcer as benign with practical certainty while differentiation between a penetrated or calloused benign ulcer and a small ulcerated carcinoma may be impossible roentgenologically. While there are unquestionably gradations between simple and penetrating ulcers, the distinction is nevertheless sufficiently clear-cut to be of considerable usefulness. The roentgen features or criteria of a benign ulcer, to be described below, may be used for all practical purposes as indicative of a simple chronic peptic ulcer.

The literature also contains conflicting reports[5,8] on the relative frequency of benign gastric ulceration in the various portions of the stomach. The results are likely to be different in resected specimens from those obtained at autopsy and both are likely to differ from series which include patients who do not come to operation or autopsy. The relative frequency of ulceration of the prepyloric portion of the stomach at autopsy[10] appears to be higher than in operative specimens. Moreover, Palmer[9] reported that small gastric ulcers, *i.e.*, ulcers not greater than 1 cm. in diameter, were most common in the pyloric and antral regions.

Over a period of years, we developed several clinical impressions about prepyloric benign ulcers—namely; (1) that they were considerably more common than much of the literature indicated; (2) that, in the large majority of instances, prepyloric ulcers were small and presented typical features of a benign ulcer; and (3) that when such features were present, the lesion could safely be called benign without reservation. Despite the fact that such cases were reported to be benign without equivocation, the possibility of carcinoma was usually permitted to sway clinical judgment. In several instances, resection was performed, despite the facts that the patient appeared to respond well to medical treatment and the pathologist had considerable difficulty in identifying any ulceration at all. These lesions had healed with little residue. In order to substantiate our clinical impressions, it was decided to review patients hospitalized over a five year period with benign ulceration of the prepyloric segment and with carcinoma in this area.

ROENTGEN CRITERIA

In conformity with others, the prepyloric segment of the stomach was considered to extend for an inch proximal to the pyloric sphincter. The concept of this segment as a separate unit is based on functional studies indicating that this segment acts as a distinct unit. Ulcerations located directly on the pyloric sphincter were not included as prepyloric ulcers since it was often uncertain whether these were duodenal or gastric.

Before presenting the results of the review of cases, it is necessary to indicate the roentgen features or the criteria which were used to arrive at the diagnosis of a benign prepyloric ulcer. Since these criteria were established after a preliminary review of the material, the fact that they fit the group of cases studied does not prove that they are necessarily the most suitable. Nevertheless, it was necessary to establish criteria in order to determine what fraction of the cases of benign ulceration in the prepyloric segment would thereby be included. Obviously, if the criteria were so stringent as to be useful in only a small percentage of cases, they would be of no great practical value. As demonstrated below, however, the criteria selected were fulfilled by all benign simple ulcers in this area. Moreover, they were not fulfilled in any case of carcinoma of the same region. It should not be assumed, however, that if an ulceration in the prepyloric portion of the stomach does not fulfill these particular criteria, it is necessarily malignant. There remains a group of lesions in which features of both benign ulceration and ulcerated carcinoma are present. Despite the fact that the weight of evidence may point in



Fig. 1. A small benign simple prepyloric ulcer is present along the lesser curvature with the typical appearance of a niche (arrow). A long neck is present, located in the center of a rather discrete ulcer mound. This pseudodefect is sharply demarcated and smooth in contour. A thick vertical fold extends inferiorly from the site of ulceration.

one or another direction in a particular case, the diagnosis must be considered to be indeterminate. In a third group of cases, the roentgen findings are unequivocal of carcinoma.

The criteria used to indicate that a simple benign prepyloric ulceration was present were the following: (1) A single discrete ulcer (Fig. 1) is clearly demonstrated within the prepyloric portion of the stomach. The major difficulty with this criterion is differentiation from duodenal ulcers at the base of the bulb. Cases were selected for study only if the position of the pylorus was clearly defined and the ulceration could be placed on the gastric side of the pyloric sphincter. (2) The ulcer is located along or near the lesser curvature. (3) The ulceration does not exceed 1 cm. in maximum diameter. (4) The mucosal pattern about the crater is intact (Fig. 2). In the case of ulcers that do not present as niches, radiation of folds to the edge of the crater is a necessary criterion (Fig. 3, *A–D*). When the ulcer presents as a niche, radiation of folds is sometimes not clear-cut but it must be evident that the fold pattern in immediate relationship to the crater is still present (Fig. 4, *A* and *B*). The folds usually appear somewhat thickened but the valleys between the folds are thin and sharply outlined. (5) Generalized spasm or persistent diffuse deformity of the prepyloric segment is absent. Cases in which the prepyloric segment was reduced to a narrow channel were excluded. This criterion can be phrased in another fashion —namely, that motor activity of the prepyloric segment in the nature of intermittent contraction is maintained. At some

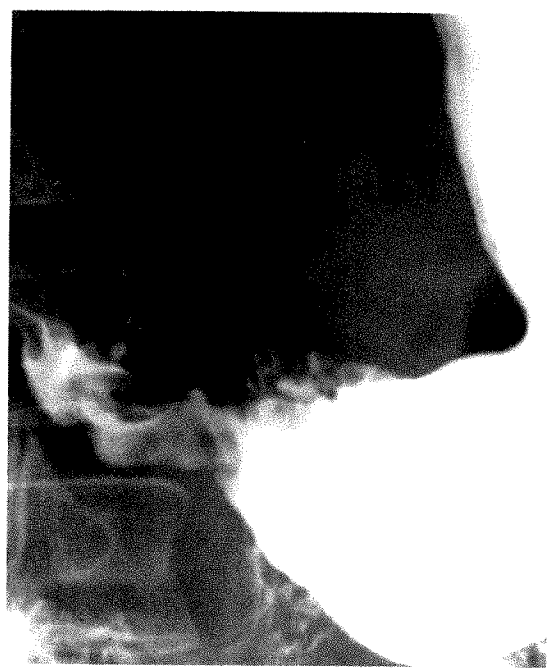


Fig. 2. A simple prepyloric ulcer appears as a niche along the lesser curvature. A rather clear-cut radiating pattern of folds extends to the edge of the crater. The folds are thick but the valleys between the folds are relatively thin and sharply outlined. The gastric wall around the ulcer crater is flattened but there is no angulation or sharp demarcation from the adjacent normally distensible stomach wall. While the antrum never distended in completely normal fashion, the configuration of this portion of the stomach was not bizarre and intermittent contraction resembling normal motor activity of this portion of the stomach was present.

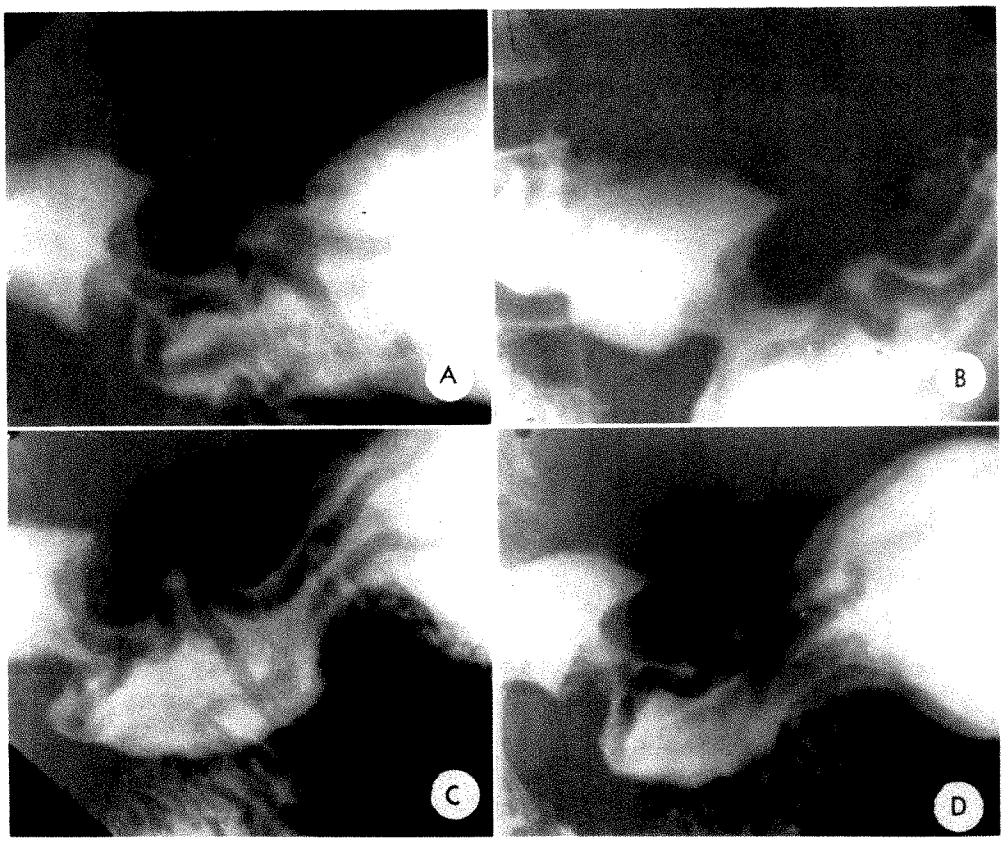


FIG. 3. (*A*, *B*, *C* and *D*). Serial roentgenograms on a polygraph of the prepyloric portion of the stomach showing a simple prepyloric ulcer. The patient is prone. The crater does not have the appearance of a niche. The varying fold pattern is well demonstrated. Exquisite radiation of the folds to the edge of the ulcer crater is evident only during certain phases of motor activity. The ulcerated area also participates in the motor activity of the region so that the crater is not always filled.

phase of filling or emptying of the prepyloric segment, the characteristic configuration of this portion of the stomach must be recognizable. (6) In only a rare case is there normal distensibility of the stomach wall about an ulcer crater. In practically every instance, there is an ulcer collar or mound[14] surrounding the crater which presents as a pseudofilling defect. For inclusion as a simple benign ulcer, it is required that this localized area of flattening or area of lack of distensibility be sharp and smooth in contour with the ulceration located in its central portion. Moreover, the ulceration associated with such an area of limited distensibility must have the typical appearance of a niche.[14] The "niche" appearance is good evidence that a rigid mass is not present (Fig. 5) since it indicates a saddle-shaped deformity rather than a hemispheric filling defect. This type of deformity is the basis of the "niche sign" and is the result of spasm of the muscularis mucosae and propria around the ulcer.

MATERIAL

The roentgen reports on all patients hospitalized in The Mount Sinai Hospital from 1953 to 1958 in whom a discharge diagnosis of gastric ulcer had been recorded were reviewed. An attempt was made to re-examine the roentgenograms of all cases in which the ulcer was located distal to the re-entrant angle. In the same fashion, all patients in whom the discharge diagnosis was carcinoma of the stomach

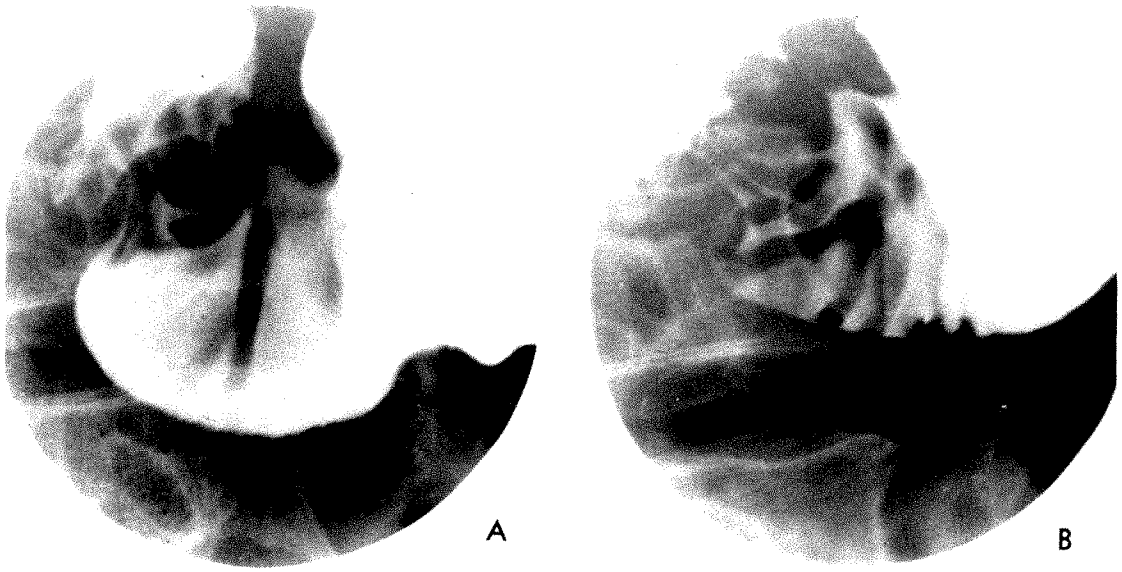


FIG. 4. (*A* and *B*) Serial spot roentgenograms with compression of the prepyloric area show a simple prepyloric ulcer with intact mucosal pattern. The mucosal folds were somewhat thickened and in the region of the crater were not completely effaced during distention. Note that the fold pattern is not stereotyped but changes in configuration presumably as a result of its own motor activity as well as contraction of the muscularis propria.

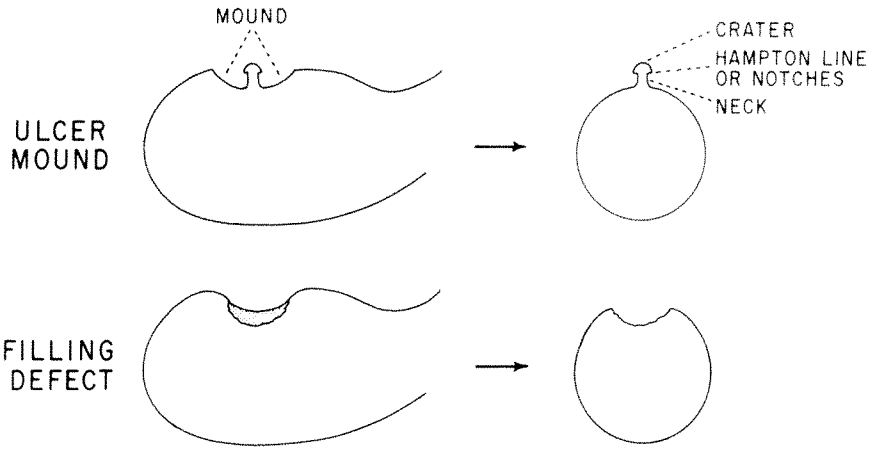


FIG. 5. Diagrammatic representation of the distinction between an ulcer mound and a filling defect. In the conventional frontal plane, both appear as defects in the contour. However, in cross section, a true filling defect is relatively symmetric while the lack of distensibility surrounding an ulcer crater is not evident as a defect. Spasm of the muscle around the crater creates a "saddle" type of deformity, convex in one plane and concave in another. For this reason, a benign crater in profile clearly projects from the contour as a niche. In contrast, a small filling defect has a variable appearance depending upon the degree of compression. An ulceration may be completely buried within it and evident only during compression. The profile features of a benign ulcer in addition to a neck with a soft-tissue collar around it include the Hampton line at the junction of the crater and the neck. A Hampton line is rarely seen in exquisite fashion with simple prepyloric ulceration but small notches at the same site can often be discerned.

were also reviewed and the roentgenograms in which the disease involved the distal part of the stomach were re-examined. The roentgen findings and the discharge diagnosis were correlated with available pathologic reports. In 12 instances, the microscopic slides were reviewed to confirm the fact that the lesions were truly chronic peptic ulcers and not erosions. All of these cases demonstrated that penetration of the ulceration extended at least through the muscularis mucosae and that the microscopic appearance of the tissue in the base of the ulcer was typically that of a peptic ulcer.

In the period of survey, there were 141 patients hospitalized and examined in the radiologic department who had a discharge diagnosis of benign gastric ulcer. In 51 of these cases, the distal portion of the stomach was involved but roentgenograms were available for re-study in only 45. Of these 45 cases, there was marked narrowing of the prepyloric segment in 4, in 6 cases differentiation from duodenal ulcer was uncertain, and in 3 cases the ulcers were so large as to extend beyond the prepyloric segment. In one case, a small ulcer was located proximal to the prepyloric segment but distal to the re-entrant angle. These 14 cases were therefore not included as examples of simple prepyloric peptic ulcer. Five additional cases were excluded from any further analysis because there was no adequate follow-up. All other cases were either confirmed by pathologic examination or during a course of follow-up which extended for at least twelve months. Twenty-six cases were therefore available for detailed study. In each case, review of the roentgenograms showed that the criteria for benign ulceration were fulfilled.

RESULTS OF ANALYSIS

The results indicate that in the hospital population, small benign ulcers in the prepyloric portion of the stomach constitute at least 25 per cent of the total number of gastric ulcers. The relative rarity of large ulcers in the prepyloric portion appears to be confirmed. Benign ulcers confined to the region between the prepyloric segment and the re-entrant angle are surprisingly uncommon.

During the same period, 160 hospitalized patients with carcinoma of the stomach were examined in the radiologic department. Eighty-four of these involved the distal portion of the stomach. In none were the criteria for simple benign prepyloric ulceration (Fig. 6, *A* and *B*) fulfilled.

The size of the ulcers on roentgen examination varied from 2 to 10 mm. The average and the median sizes were both 5 mm. Twenty-two of the ulcers were ovoid or elliptic, while 4 were somewhat rectangular. In 20 instances, the ulcers were visualized as niches projecting clearly from the lesser curvature. In 6 cases, the ulcers were seen only *en-face*. Table 1 indicates the location of the ulcers in relationship to the re-entrant angle and the pyloric ring. They appear to be rather uniformly distributed throughout the prepyloric segment. Definite radiation of folds to the ulcer crater was seen in 20 instances. In all cases, there was some enlargement or distortion of the mucosal fold pattern. In 2 cases, this was quite marked and associated with excessive secretions. In 5 instances, there was evidence of prolapse of gastric folds into the base of the duodenal bulb. In 2 instances, there appeared to be rather typical hypertrophy of the pyloric

TABLE I

LOCATION OF ULCERS

Distance from Pyloric Ring (cm.)	No. of Cases	Distance from Re-entrant Angle along Lesser Curvature (cm.)	No. of Cases
Just proximal	5	3.0	2
1.0	6	4.0	3
1.5	8	4.5	5
2.0	6	5.0	3
2.5	2	5.5	8
		6.0	5
		6.5	1

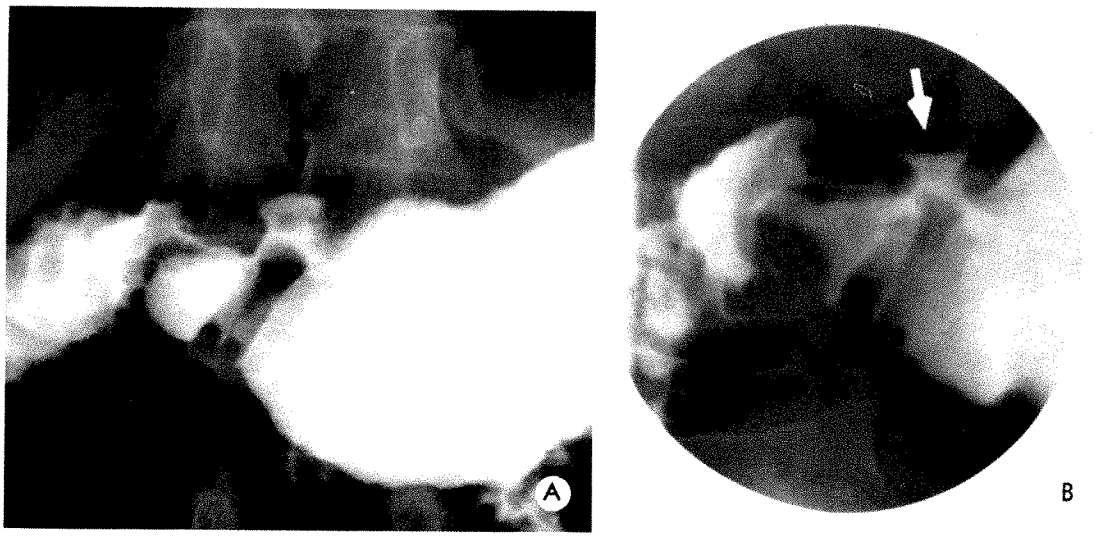


FIG. 6. A small ulcerating carcinoma of the prepyloric portion of the stomach. (*A*) A rather flat crater (arrow) is present along the lesser curvature in the center of a poorly demarcated defect. This roentgenogram shows the maximum amount of filling which was obtained in the prepyloric area throughout the course of the examination. The normal configuration of the stomach in this area was not seen at any time and typical motor activity was not present. (*B*) Spot roentgenogram with compression in the erect position shows the ulceration (arrow) with a fluid level. Several folds appear to radiate to the edge of the ulcer. However, a rolled edge which indents the crater is present along the lesser curvature at the distal margin of the ulceration. The folds distally along the lesser curvature stop abruptly at the margin of this rolled edge. A persistent bizarre configuration of the prepyloric segment plus evidence of a rolled edge along one border violate the criteria of a simple benign ulcer. This case was selected for illustration because, in the series studied, it came closer than any other to fulfilling the criteria described in the text for simple benign ulceration.

musculature. In one of these cases, confirmation was obtained at operation. In 5 instances, there was deformity of the duodenal bulb characteristic of a duodenal ulcer. There was one case with a second gastric ulcer on the posterior wall of the body of the stomach.

CLINICAL FEATURES

The clinical features in the group of 26 patients were analyzed within the limitations of the information available. The age of these patients varied between thirty-nine and eighty-three. The average and the median ages were both fifty-seven. There were 15 females and 11 males. Twenty-four patients were white and 2 were Negroes. In 2 cases, ulceration was associated with steroid therapy. Epigastric pain was present in 23 of these patients. In 6 patients, pain had been present intermittently over a period of more than five years. In 8 patients, pain had been present from one to five years. In 17 instances there was a clear history of relief by food. Frequent vomiting was a symptom in 6 instances. Weight loss, however, was a prominent feature in only 2 patients. Gross gastrointestinal bleeding was noted in 12 cases, while anemia with positive guaiac stools was present in 2 patients. One case developed a free perforation. Free acid was present in 16 cases, *i.e.*, in all cases in which the test was recorded. Pyloric obstruction was not a prominent feature in any case. In general, the symptoms did not appear to vary significantly from those of a duodenal ulcer. The fact that bleeding was a frequent feature is to be expected since this series included only hospitalized patients. It is rather surprising, however, that massive hematemesis

may result from ulcerations which have not penetrated into the serosa.

FOLLOW-UP ROENTGEN STUDY

In 8 cases, follow-up roentgenograms were available. In 2 patients, an ulcer was still present, somewhat smaller in one and unchanged in the other, seventeen and twenty-seven months respectively following the original examination. A tiny niche-like projection may persist after healing because of wrinkled folds overlying the scarred area which prevent complete distention and stretching of the mucosa (Fig. 7, *A* and *B*). In no case was there complete restoration of the prepyloric segment to an absolutely normal appearance. In some, there was only a hardly noticeable flattening or lack of distensibility of a small region of the lesser curvature, while in others a thick transverse fold or incisura-like defect remained (Fig. 8, *A* and *B*, and 9, *A* and *B*). In several cases, the persistent lack of distensibility over a short segment associated with a flattened or distorted mucosal pattern created a picture which was more difficult to differentiate from carcinoma than the original lesion (Fig. 10, *A* and *B*). It is likely that many benign antral deformities are the result of a healed prepyloric ulcer. While no cases of recurrent ulceration were noted in this group of patients, we have seen several instances of healing of a prepyloric ulcer followed by recurrence in the same area sometimes on repeated occasions. This sequence of events is quite comparable to that seen so frequently in patients with duodenal ulcers.

DISCUSSION

The roentgen features of benign ulceration and of carcinoma in the prepyloric segment of the stomach do not differ essentially from those in other areas of the stom-

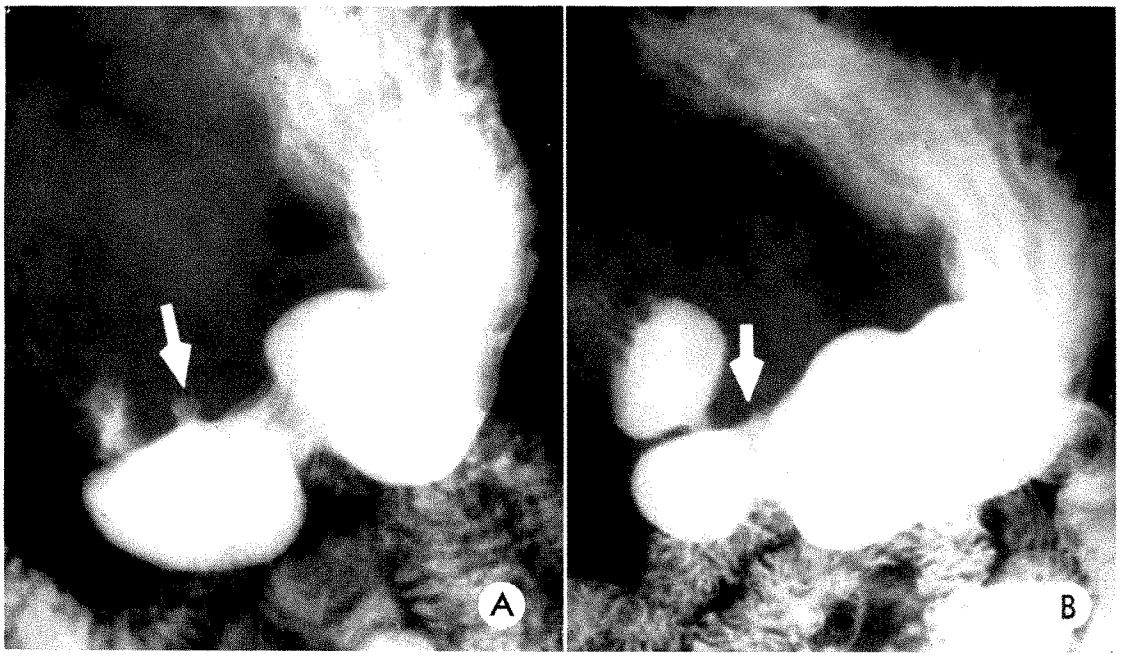


FIG. 7. (*A*) A simple prepyloric ulcer (arrow) is present along the lesser curvature which presents as a niche in the center of a flattened area. Intact radiating folds can be faintly seen extending to the edge of the ulcer. (*B*) Roentgenogram five months later shows a tiny niche-like projection (arrow) at the site of the ulceration. At this stage, it is difficult to distinguish between barium caught between distorted folds and residual ulceration. The folds adjacent to a healed scarred area persist despite maximum distention. A wrinkling of the contour with a slight diminution in the density of the barium in the area results.

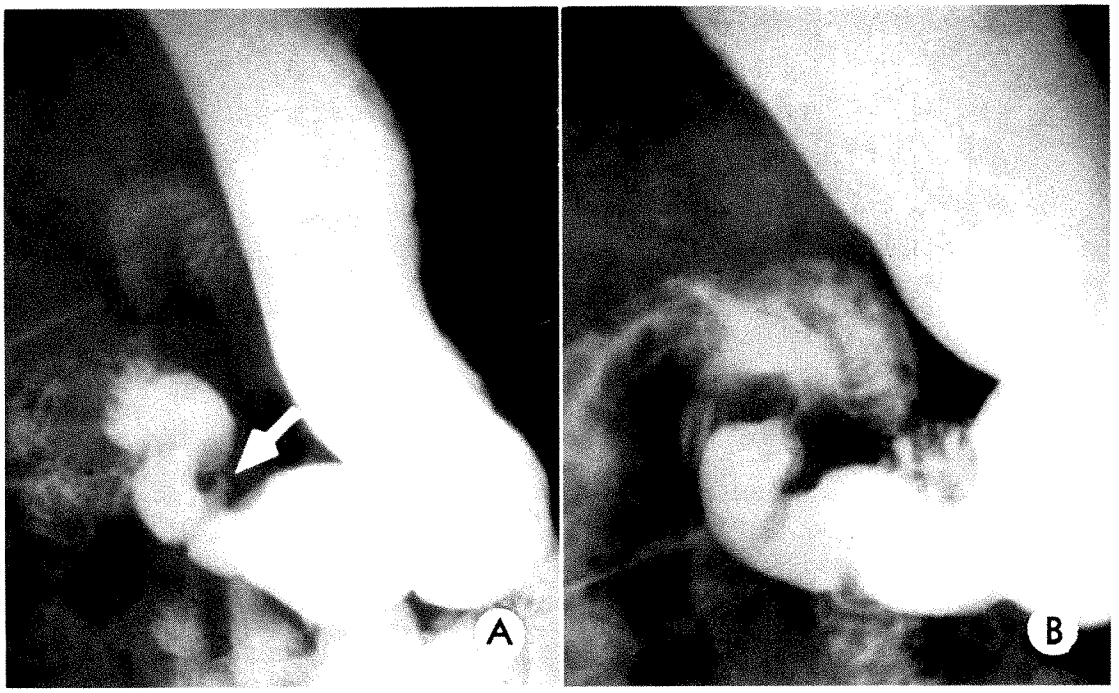


FIG. 8. (*A*) A simple prepyloric ulcer shows a typical niche appearance (arrow) within a rather discrete ulcer mound. A Hampton line is faintly seen. A thick vertical fold connects this area with the greater curvature. (*B*) Roentgenogram four months later shows no definite evidence of an ulcer crater but an incisura-like defect persists at the site of previous ulceration. The thick vertical fold is still present and is not effaced during maximum distention. The lesser curvature is shortened at the site of the incisura, as evidenced by the turning up of the stomach distal to this point.

ach.[1,12] This segment has received special attention only because it has been considered that the conventional criteria are misleading in this region. The distal portion of the stomach is often easier to examine than the remainder of the stomach because of its smaller size and the ability to apply graded compression more conveniently. Moreover, the mucosal pattern in this area of the stomach is ordinarily simple to demonstrate and constant in configuration, and motor activity is more pronounced than elsewhere in the stomach. Small benign ulcers, a few millimeters in diameter, appear to be unusually frequent in this area as compared with the remainder of the stomach, but this may be due to limitations of the method of examination in other areas. It is true that, more commonly than elsewhere in the stomach, benign ulceration of the prepyloric portion of the stomach may be associated with marked spasm and perhaps gastritis. The roentgen features of the ulceration *per se* may therefore be more difficult to delineate. Similar spasm may be produced by a malignant ulcer or an ulcerated carcinoma or an infiltrating scirrhous carcinoma. As pointed out above, when diffuse or bizarre narrowing is present, an unequivocal diagnosis of a simple benign prepyloric ulcer cannot be made.

It is becoming general practice for the radiologist to report ulcerated lesions of the stomach as benign, indeterminate or malignant.[10,11,12] The indeterminate group actually consists of two subgroups; namely, one in which the examination is unsatisfactory because of excessive secretions or bizarre motor activity and a second in which the roentgen features do not all point in the same direction. This is of considerable assistance in evaluating the accuracy of roentgen reports.

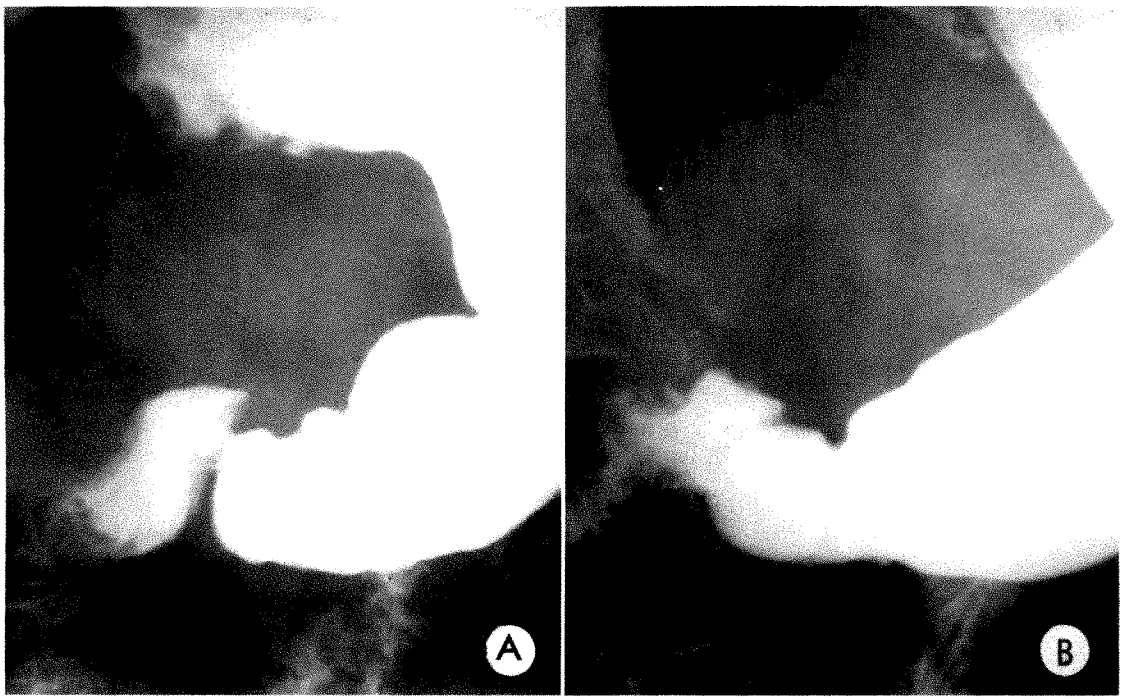


Fig. 9. (*A*) A simple prepyloric ulcer is present along the lesser curvature with an atypical niche appearance. There is no neck to the crater. (*B*) Roentgenogram one year later shows no evidence of ulceration but a small incisura or a thick fold is present at the same site.



Fig. 10. (*A*) A small prepyloric ulcer is present in the center of an ulcer mound. The fold pattern is intact and extends to the edge of the crater although radiation of folds is not evident. (*B*) Roentgenogram four months later shows that the ulceration has healed but there is a localized area of decreased distensibility. On compression studies, however, the fold pattern in this area appeared to be intact and the configuration changed during different phases of filling. The patient has remained well for a period of four years.

The results of the study described above may be legitimately criticized on the basis that it was started with preconceived ideas and conducted with personal bias. It must also be admitted that the results may be coincidental to some extent since it is likely that few small malignant ulcers will be represented in any series of symptomatic patients. There is no question that, with additional experience, a malignant prepyloric ulcer will be found which fulfills all of the criteria of a simple benign ulcer roentgenologically and on gross pathologic examination. Nevertheless, the practical fact is that we have had no difficulty clinically. Carcinomas of the stomach have not been overlooked by using the criteria described. This statement applies not only to the hospitalized series of cases studied in this report but to a considerably larger group of patients seen and followed on an ambulatory basis by ourselves and our colleagues. Another criterion of the benign nature of a gastric ulcer in the prepyloric segment of the stomach or elsewhere, however, should be added. This is essentially a clinical criterion; *i.e.*, a test of treatment. In every case, it is recommended that close supervision and intensive medical care be practiced until the ulcer is completely healed.[7] A recommendation for re-examination after a stated interval of time is included in the radiologist's report. One cannot, however, insist that the roentgen appearance of the stomach return completely to normal, since some deformity usually persists. It should be possible to demonstrate that the abnormality is simply a deformity; that is, no defect or rigid area remains and an intact fold pattern is present. In any particular case, the decision for or against operative intervention should be made in a fashion similar to that with duodenal ulcer, on the basis of the clinical course of the patient rather than the fear of overlooking a carcinoma.

SUMMARY

1. It is not true that the majority of prepyloric ulcers are malignant. On the contrary, simple ulcers of this area are overwhelmingly benign.

2. From the point of view of the roentgen differential diagnosis of prepyloric ulcers, it is important to distinguish between simple, nonpenetrating ulcers and penetrating calloused ulcers. A correct roentgen diagnosis is much simpler in the first group.

3. The roentgen criteria of simple peptic ulceration of the prepyloric portion of the stomach are adequate to exclude the possibility of carcinoma for all practical purposes.

4. The roentgen findings and the clinical course of patients with simple prepyloric gastric ulceration are not greatly different from those of patients with duodenal ulcer. It is common for a deformity to persist after healing of a benign gastric ulcer.

Bernard S. Wolf, M.D.
The Mount Sinai Hospital
11 E. 100 Street
New York 29, New York

We should like to acknowledge the assistance of the Department of Pathology, specifically Dr. Ken Mori, for review of the microscopic slides.

REFERENCES

1. ABBOT, F., and BLANK, S. Gastric ulcer—benign or malignant. *New England J. Med.*, 1953, *249*, 722–726.
2. COLLINS, J. A. Analysis of pyloric and prepyloric lesions. *Pennsylvania M. J.*, 1952, *55*, 331–332.
3. DOUB, H. P. Differential diagnosis of pyloric and prepyloric ulcerations. AM. J. ROENTGENOL. & RAD. THERAPY, 1940, *43*, 826–831.
4. HAMPTON, A. O. Incidence of malignancy in chronic prepyloric gastric ulcerations. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 473–479.
5. HELLWIG, C. A., WELCH, J. W., and WILKINSON, P. N. Localization of stomach ulcers. *A.M.A. Arch Surg.*, 1958, *76*, 331–333.
6. KIRKLIN, B. R., and MACCARTY, W. C., JR. Incidence of malignancy in prepyloric ulcers. *J.A.M.A.*, 1942, *120*, 733–735.
7. KIRSNER, J. B., CLAYMAN, C. B , and PALMER, W. L. Problem of gastric ulcer. *A.M.A. Arch. Int. Med.*, 1959, *104*, 995–1020.

8. OI, M., OSHIDA, K., and SUGIMURA, S. Location of gastric ulcer. *Gastroenterology*, 1959, *36*, 45–56.
9. PALMER, E. D. Clinical significance of small benign gastric ulcer, with note on benign ulcer of greater curvature and in absence of free hydrochloric acid. *Am. J. M. Sc.*, 1952, *223*, 386–391.
10. PORTIS, S. A., and JAFFE, R. H. Study of peptic ulcer based on necropsy records. *J.A.M.A.*, 1938, *110*, 6–13.
11. SCHWARZ, G. S. X-ray differentiation between benign and malignant gastric lesions. *Gastroenterology*, 1950, 15, 67–74
12. SCOTT, W. G., LOITMAN, B. S., and SWANSON, H. A. Problems for radiologist in diagnosis of gastric ulcer. *J.A.M.A.*, 1959, *171*, 2048–2053.
13. SINGLETON, A. C. Benign prepyloric ulcer. *Radiology*, 1936, *26*, 198–208.
14. WOLF, B. S., and MARSHAK, R. H. Profile features of benign gastric niches on roentgen examination. *J. Mt. Sinai Hosp.*, 1957, *24*, 604–626.

THE ROENTGEN APPEARANCE OF SPONTANEOUS INTRAMURAL HEMORRHAGE OF THE SMALL INTESTINE ASSOCIATED WITH ANTI-COAGULANT THERAPY*

By HYMAN R. SENTURIA, M.D., NOAH SUSMAN, M.D., *and* HERMAN SHYKEN, M.D.
ST. LOUIS, MISSOURI

THE use of anticoagulant therapy in the management of diseases associated with occlusive vascular changes has occasionally given rise to complications due to the defective clotting mechanism. Since patients with some of these diseases, such as cerebral or coronary thrombosis, are poor surgical risks, it is imperative to recognize that the clinical findings suggesting an acute surgical abdomen may be secondary to intramural or intramesenteric bleeding of the intestine, a condition often lending itself to nonoperative clinical management. Spencer, Bateman and Horn,[6] in their review of 23 cases of intramural hematoma which caused small intestinal obstruction, observed that in more than two-thirds of the cases there was a history of antecedent trauma. In 2 cases, a definite bleeding tendency was observed; one a hemophiliac, the other a newborn infant with an undiagnosed hemorrhagic diathesis. No mention was made of the association of anticoagulant therapy with any of the reported cases. The first report of spontaneous intramural hemorrhage of the small bowel associated with anticoagulant therapy was made by Berman and Mainella[1] in 1952. They reported 5 cases presenting with overt hemorrhage. Of these, 3 had findings of an acute surgical abdomen. Mendelsohn and Iglauer[4] reported 2 cases of spontaneous intraperitoneal bleeding due to dicumarol intoxication. Single cases of spontaneous localized intramural hemorrhage with surgical resection of the involved small intestinal segment occurring in patients under anticoagulant therapy were described by Pearson and MacKenzie[5] and by Gilbert and Jorgenson.[3]

No contrast studies of the small intestine with intramural hemorrhage resulting from anticoagulant therapy have been reported. The 3 cases to be described in this report were studied with barium and a consistent and characteristic roentgen appearance was demonstrated. The roentgen findings in the small intestinal study in the first case were correlated with the anatomic changes found during surgery and were sufficiently typical to suggest the correct roentgenologic diagnosis in the 2 subsequent cases. Characteristically, the findings are those of a rigid segment of small intestine with an intact coarsened mucosal pattern. The lumen is diminished and narrow spike-like projections of barium extend to outline the normal width of the bowel. The appearance mimics an infiltrative process of the bowel wall.

REPORT OF CASES

CASE 1. A sixty year old white male was admitted to the Jewish Hospital on September 10, 1957, with the chief complaint of diffuse abdominal pain of two days' duration. Three months earlier, he had been hospitalized because of left sided headaches, right homonymous hemianopsia, and right hypesthesia secondary to a carotid artery occlusion. Dicumarol therapy was initiated and maintained after the patient was discharged from the hospital. Prothrombin times were checked weekly, but three days prior to this admission the patient failed to keep his laboratory appointment. Two days before admission the patient complained of low midabdominal pain which then became generalized and was accompanied by nausea and vomiting. The symptoms became progressively more severe. He had had no bowel movements for the twenty-four hours preceding hospitalization. The physical examination

* From the Division of Radiology, The Jewish Hospital, St. Louis, Missouri.

showed a well developed, well nourished male in acute abdominal distress, lying supine with his legs drawn up. The blood pressure was 160/100, the pulse rate 100 and regular, and the temperature 100° F. The skin showed no ecchymoses. The abnormal physical findings were limited to the abdomen. There was no distention. The bowel sounds were absent. There was diffuse muscle guard, more marked on the right. Tenderness to rebound was present throughout. There was right costovertebral angle tenderness. Laboratory examination revealed a hemoglobin of 14.7 gm. per cent and a white blood cell count of 12,900. The prothrombin time was 5 per cent of normal, and the serum diastase was 120 Somogyi units. Urinalysis, including microscopic examination of the sediment, was negative.

The patient was thought to have an acute surgical abdomen, but operation was postponed because of the low prothrombin time. The patient was given 50 mg. of mephyton intravenously. A Levine tube was passed and connected to continuous suction and wide spectrum antibiotics were started. Five hours later, the hemoglobin had dropped to 11.4 gm. per cent and the prothrombin time had increased to 36 per cent of normal. A fleck of stool on the rectal examining finger was hemotest positive. On the second hospital day, the abdomen continued to be rigid and diffusely tender and the temperature rose to 102° F. The white blood cell count was 13,500 and the prothrombin time had risen to 70 per cent. The patient began to improve on the third day, showing less rigidity and some return of bowel sounds, but continued to show diffuse abdominal tenderness. The patient was transfused with one unit of blood. The white blood cell count had dropped to 8,000. The patient continued to improve, and on the fourth day the Levine tube was removed. On the fifth day, the patient passed two tarry stools. Antibiotics were discontinued on the sixth day, and the patient became afebrile. The tenderness subsided on the seventh day, and the patient was now asymptomatic. On the tenth hospital day, a barium study of the small intestine showed a segment of proximal jejunum approximately 30 cm. in length with a narrowed lumen, rigid wall and coarsened edematous valvulae conniventes (Fig. 1). Multiple diverticula and atypical spike-like projections of barium outlining the normal caliber of the bowel lumen were present. Because of the findings of diverticula together with an apparent infiltrating lesion of the bowel wall, a diagnosis of diverticulitis of the jejunum was made and surgery was advised. At operation, a moderate amount of old blood was present in the peritoneal cavity. A segment of the small intestine (Fig. 2) about one foot distal to the ligament of Treitz showed a number of very large diverticula and diffuse hemorrhage into the bowel wall. This segment of small intestine was resected. Microscopic examination showed the mucosa to be intact (Fig. 3). Within the submucosa, there was widespread hemorrhage with no cellular reaction. There was moderate edema and hemorrhage throughout all layers. The pathologist's diagnosis was intramural hemorrhage of the small bowel with localized jejunal diverticulosis.



FIG. 1. Case 1. Examination of the small intestine one hour after the barium meal shows in a segment of the proximal jejunum (arrows) the characteristic roentgen appearance of intact mucosa, coarsened edematous folds and spike-like projections extending out to the normal luminal caliber. Note the presence of diverticula in this segment. The barium in the colon is from a previous barium enema study.

Comment. The presence of diverticulosis of the jejunum was a misleading factor, and the striking serrated appearance of the involved segment of the small intestine in an area of extensive diverticulosis led to an incorrect diagnosis of diverticulitis of the jejunum. Even though the patient had im-

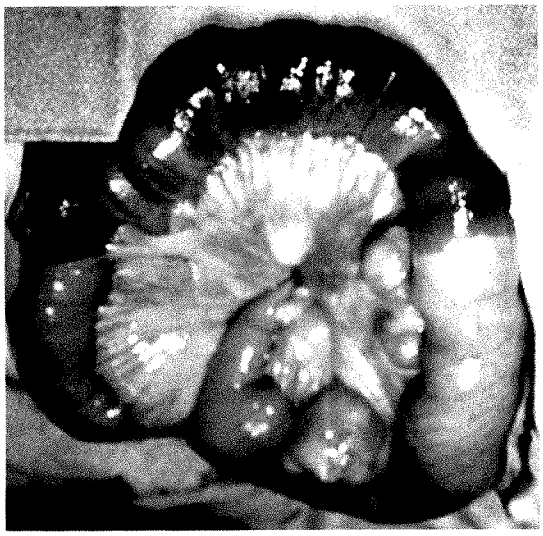


FIG. 2. Case 1. A photograph of the surgical specimen showing the sharply demarcated hemorrhagic segment.

proved markedly on a conservative regimen, surgery was decided upon because of the bizarre roentgen appearance in the jejunum. It is interesting to speculate that the presence of diverticula in the jejunum may have contributed to the localization of the massive hemorrhage to this portion of the small intestine.

CASE II. A sixty-seven year old Negro female entered the Jewish Hospital for the second time on June 8, 1959, complaining of fever and abdominal pain. Four months earlier, the patient was admitted to the hospital because of myocardial ischemia. She was started on dicumarol therapy which was continued after discharge. Five days prior to this admission, the patient began having chills, fever and abdominal pain. This was soon followed by abdominal distention, nausea and vomiting. The prothrombin time was at a therapeutic level. Subsequently, the temperature rose to 103° F., and the distention increased. She became progressively worse, and one day prior to admission she was unable to retain anything by mouth. The patient gave the history of having had an appendectomy thirty years previously. On physical examination, the blood pressure was 120/68, the pulse rate was 120 and regular, and the temperature was 101° F. The skin showed no hemorrhages or petechiae. The positive physical findings were limited to the abdomen. There was distention and the bowel sounds were hypoactive. The entire abdomen was tympanitic to percussion except in the flanks. There was generalized tenderness with moderate rebound tenderness throughout. There was a vague right upper quadrant mass. The laboratory examination revealed a hemoglobin of 8.8 gm. per cent and a white blood

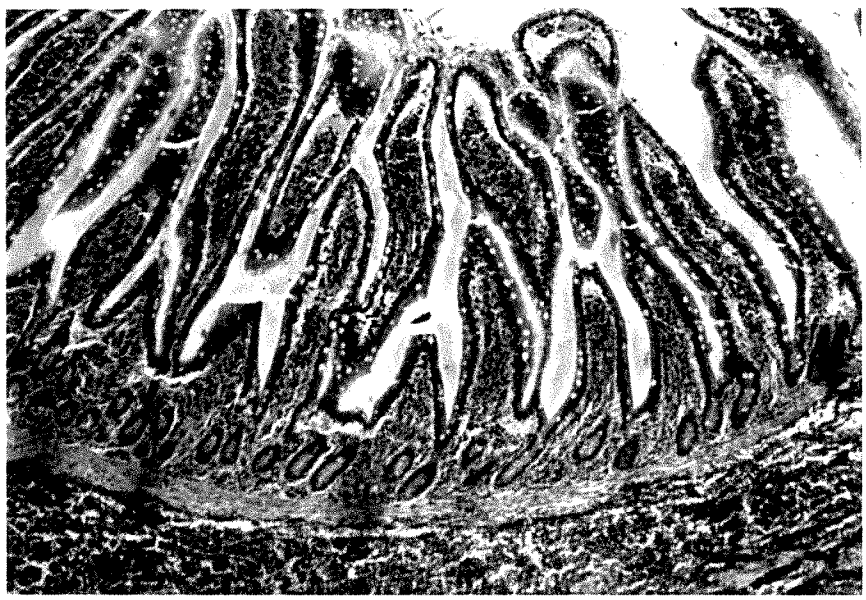


FIG. 3. Case 1. A photomicrograph of the wall of the resected bowel showing an intact normal appearing mucosa and diffuse submucosal hemorrhage.

cell count of 17,600 with 33 per cent stabs. The urinalysis was negative. Roentgen examination of the abdomen revealed gas in numerous loops of small bowel and also throughout the colon. A diagnosis of adynamic ileus was made. On the second hospital day, the patient's temperature rose to 103° F. Abdominal distention continued with hypoactive bowel sounds. The hemoglobin dropped to 6.8, then to 6.0 gm. per cent. The white blood cell count rose to 19,100, and then to 21,300 with 48 per cent stabs. The prothrombin time was markedly prolonged. Fifty milligrams of mephyton were given intravenously, and two units of blood were administered. A barium enema examination showed a normal colon without evidence of obstruction. Because the postevacuation roentgenograms showed complete emptying of the large intestine, the intestinal tonus was thought to be normal. Due to the presence of continued distention of the small intestinal loops, the diagnosis was changed to incomplete mechanical small intestinal obstruction. A barium study of the small intestine (Fig. 4) showed a narrowed rigid segment of proximal jejunum. There was considerable coarsening of the valvulae conniventes. Sharp spike-like projections of barium extended between coarsened mucosal folds to outline a lumen of normal size. The small intestine distal to this area was distended. A diagnosis of intramural hemorrhage of the proximal jejunum was made as well as small intestinal obstruction at a more distal point. Since the hemoglobin had risen only to 8.2 gm. per cent, two more units of blood were administered. The white blood cell count remained at 20,400. On the fourth hospital day the temperature rose to 104° F., the hemoglobin to 12.5 gm. per cent and the prothrombin time was 90 per cent of normal. Because there was no improvement in the patient's clinical condition, a laparotomy was performed. At operation, a perforated appendix with localized abscess formation was found and removed. A 15 cm. segment of the jejunum starting at the ligament of Treitz was thickened and deeply hemorrhagic. The mesentery was also hemorrhagic. Approximately 18 inches distal to this segment there was another hemorrhagic segment, not as severely involved as the first, which was resected. On cut section, the wall of the specimen was markedly thickened with hemorrhage in all layers of the intestine and in the mesentery. Grossly, the mucosa appeared intact. Microscopic examination revealed hemorrhage permeating all the layers of the bowel dissecting an intact mucosa from the submucosa. The appendix was acutely inflamed and perforated. On the first postoperative day the patient became afebrile and, except for a wound infection, made an uneventful recovery. Nineteen days later a repeat barium study of the small intestine showed the proximal jejunum to be normal (Fig. 5). The lesion previously demonstrated on roentgenograms and proved at surgery had shown complete anatomic reversibility. Another examination six months later again revealed no jejunal abnormality.



Fig. 4. Case 11. Roentgenogram of the gastrointestinal tract at one hour after the barium meal showing the long rigid infiltrated appearing segment of the most proximal jejunum (arrows). The small intestine is greatly distended, indicative of an obstruction at a more distal point.

Comment. In this case an acute appendicitis with perforation and localized abscess formation was complicated by a spontaneous intramural hemorrhage into two areas of the jejunum. It is possible that peritonitis secondary to the perforated appendix may have been a factor in producing a rather rapid lowering of an otherwise well controlled prothrombin level. Roentgenograms of the small intestine demonstrated extensive changes of the proximal jejunum secondary to intramural hemorrhage. This was confirmed at operation. Two follow-up roentgen examinations, made nineteen days



FIG. 5. Case II. Repeat barium study made nineteen days after Figure 4 shows a completely normal small intestinal pattern.

and again six months later, showed complete anatomic reversibility of the lesion.

CASE III. A forty-five year old white male was admitted to the Jewish Hospital for the third time on June 3, 1960, complaining of a stomach ache and bruises. Five months earlier, he was admitted to the hospital for the first time with a diagnosis of acute myocardial infarction. He was hospitalized for four weeks and started on dicumarol which was continued after discharge. One month prior to the present admission, he was hospitalized for the second time because of retrosternal pain, and was discharged six days later with a diagnosis of coronary insufficiency. The dicumarol therapy was continued throughout that admission and after discharge. Three days prior to this admission, the patient experienced the onset of nausea, aching localized in the muscles of his legs, as well as generalized myalgia. For the previous twenty-four hours he noted multiple skin ecchymoses and the onset of bilateral lower quadrant abdominal aching with increasing generalized myalgia. On physical examination, the blood pressure was 130/70, the pulse rate was 96 and regular, and the temperature was 99° F. The patient was a well developed, well nourished male with multiple ecchymoses and subcutaneous hematomas. The abdomen was slightly protuberant, and the bowel sounds were hypoactive. There was no muscle guard. There was moderate generalized tenderness and rebound tenderness more marked in the lower abdomen. Laboratory examination revealed a hemoglobin of 13.8 gm. per cent. The white blood cell count was 16,150 with 5 per cent stabs. The prothrombin time was 12.8 per cent of normal. The stool was faintly positive for occult blood. Roentgenograms of the abdomen showed several nondistended gas-containing loops of small intestine with gas and feces present in the colon. This was interpreted as indicative of an adynamic ileus. A small intestinal series was requested because of the possibility of a spontaneous intramural hemorrhage. The patient was given nothing by mouth and was administered 50 mg. of mephyton intravenously. On the evening of admission the temperature was 100.8° F.

The next day the patient's condition improved. The bowel sounds were active and now there was only minimal lower abdominal tenderness. The patient was afebrile, and the white blood cell count had dropped to 8,500. The prothrombin time was now normal. The stool was positive for occult blood. The patient continued to improve and on the fourth hospital day a barium study of the small intestine was obtained. A segment of the midileum measuring approximately 25 cm. in length showed narrowing of the lumen, coarsening of an intact mucosa and some spike-like projections of barium outlining the normal caliber of the lumen (Fig. 6, *A* and *B*). A roentgen diagnosis of spontaneous intramural hemorrhage of the small intestine was made. The patient's condition continued to improve, and he was discharged one week after admission. Two weeks after discharge, a repeat small intestinal series revealed no abnormality in the bowel pattern.

Comment. This patient, on anticoagulant therapy because of coronary occlusive disease, developed symptoms and signs of an acute surgical abdomen which responded to conservative treatment. Roentgen examination showed the characteristic changes of spontaneous intramural hemorrhage in the midileum. Complete reversibility of the process was demonstrated on roentgenograms made three weeks after the acute episode.

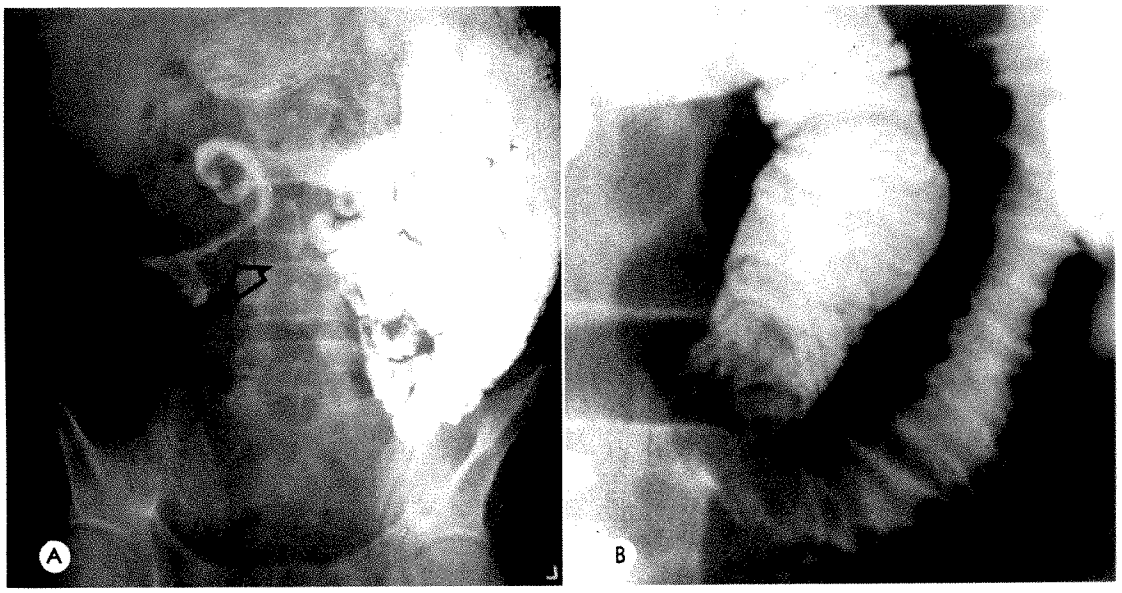


FIG. 6. Case III. (*A*) Barium study of the small intestine shows delay in intestinal motility in the distal ileum indicative of incomplete intestinal obstruction. (*B*) Detail spot roentgenogram of the abnormal ileal segment demonstrates the characteristic findings of spontaneous intramural hemorrhage.

DISCUSSION

There are many reports in the surgical and radiologic literature of intramural hematoma of the small intestine. Most of these occurred in the duodenum and were secondary to trauma. Felson and Levin[2] described the characteristic roentgen findings of traumatic intramural hematoma. They considered the presence of an intramural tumor mass, a coiled spring appearance and coarsening of the mucosal folds in the adjacent intestine pathognomonic of intramural hematoma. This paper summarizes the clinical and roentgen findings of spontaneous intramural hemorrhage in the small intestine occurring during anticoagulant therapy. The important clinical and laboratory findings from 6 of the previously recorded cases and the 3 cases reported here are presented in Table 1. These data suggest the following typical case history.

A patient on anticoagulant therapy because of occlusive vascular disease suddenly develops abdominal pain, usually cramping in nature, accompanied by nausea and vomiting and low grade fever. Physical examination of the abdomen reveals it to be distended with the bowel sounds hypoactive to absent. There is tenderness to abdominal palpation, both direct and rebound. Muscle guard may or may not be present. A vague mass may be palpable. Gross bleeding into the skin or from the gastrointestinal or urinary tract may make the hemorrhagic state obvious. A drop in hemoglobin or laboratory testing of the stool and urine may show evidence of occult blood loss. The prothrombin time is below therapeutic levels. The white blood cell count is elevated. Roentgenograms of the abdomen reveal numerous gas-distended loops of small intestine, but with gas also visualized along the course of the colon. Barium studies of the small intestine demonstrate the characteristic findings of spontaneous intramural hemorrhage. These are well demonstrated on the roentgenogram of the resected small intestinal segment from our Case II after distention with barium (Fig. 7). The appearance is that of an infiltrative process of the intestinal wall rather than that of a localized intramural tumor. The segment of small intestine is narrowed and rigid and

TABLE I

SYMPTOMS, CLINICAL FINDINGS AND LABORATORY DATA IN 9 CASES OF SPONTANEOUS INTRAMURAL HEMORRHAGE OF THE SMALL INTESTINE

		Symptoms		Findings on Physical Examination of Abdomen						Laboratory Findings						
													Evidence of Abnormal Bleeding			
													Gross		Occult	
No.	Authors	Abdominal Pain	Nausea and Vomiting	Distention	Bowel Sounds	Muscle Guard	Tenderness	Rebound Tenderness	Temperature (F)	White Blood Cell Count	Prothrombin Time	Drop in Hb	Gastrointestinal Tract	Urinary Tract	Stool	Urine
1	Berman and Mainella[1]	present (cramping)	present	present			present		102.0°			present	present		present	
2	Berman and Mainella[1]	present (cramping)	present	present			present		101.0°		30 sec.					
3	Mendelsohn and Iglauer[4]	present (cramping)	present	present	absent		present				8%			present		present
4	Mendelsohn and Iglauer[4]	present	present	present		present	present	present	99.8°	12,600	0%					present
5	Gilbert and Jorgenson[3]	present (cramping)	absent (anorexia)	absent	hyperactive		present		99.7°	8,600	11%	present			absent	present
6	Pearson and MacKenzie[5]	present (cramping)		present	absent				98.6°		L10%		present		present	absent
7	Senturia *et al.*	present		absent	absent	present	present	present	102.0°	13,500	5%	present	present	absent	present	absent
8	Senturia *et al.**	present	present	present	hypoactive	present	present		104.0°	21,300	prolonged	present	absent	absent	none reported	
9	Senturia *et al.*	present	present	present	hypoactive	absent	present	present	99.0°	16,150	12.8%		absent	absent	present	absent

* This case (our Case 11) was complicated by the presence of acute appendicitis with perforation and abscess formation which distorted the clinical picture.

shows a coarsened, intact mucosal pattern. There are occasional narrow spike-like projections of barium extending to outline the normal width of the lumen. The segment is rigid because of hemorrhage permeating through all layers of the intestine. There is narrowing of the lumen because the relatively nondistensible serosa causes the hemorrhage to expand centripetally, and therefore, encroach upon the lumen. The intact mucosa is rigid and coarsened in appearance because of the dissection of hemorrhage into the easily separable plane between the muscularis mucosae and the muscular layer.

In 2 of our cases, we have roentgenographically observed this lesion to be completely reversible and amenable to nonsurgical management. It is, therefore, imperative to recognize this condition in patients on anticoagulant therapy who may be poor operative risks due to severe coronary or cerebrovascular disease.

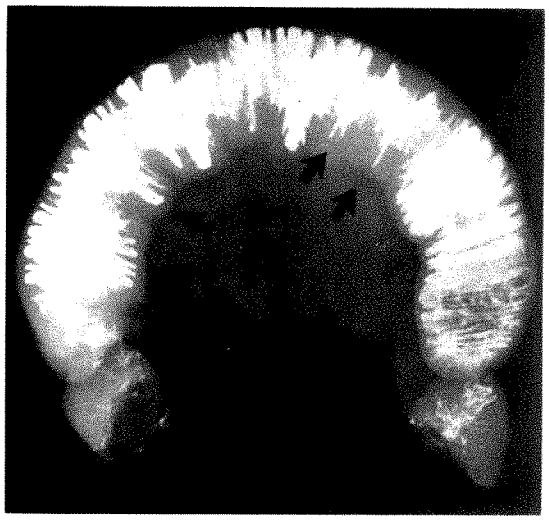


Fig. 7. Case II. Roentgenogram of the resected segment of the small intestine after intraluminal distention with barium. The mucosal thickening, the narrowing of the lumen, and several spike-like projections (arrows) are well demonstrated.

SUMMARY

1. Three cases of spontaneous intramural hemorrhage of the small intestine occurring in patients on anticoagulant therapy are reported.

2. The clinical findings in these cases and in 6 of the collected cases from the literature are presented.

3. A characteristic roentgen finding on barium study of the small intestine in these cases is described.

4. Serial roentgenograms show complete anatomic reversibility of these abnormal small bowel changes.

Hyman R. Senturia, M.D.
Suite 112, Pasteur Building
462 North Taylor
St. Louis 8, Missouri

REFERENCES

1. Berman, H., and Mainella, F. S. Toxic results of anticoagulant therapy. *New York J. Med.*, 1952, *52*, 725–727.
2. Felson, B., and Levin, E. J. Intramural hematoma of duodenum; diagnostic roentgen sign. *Radiology*, 1954, *63*, 823–831.
3. Gilbert, A. E., and Jorgenson, N. C. Small bowel obstruction due to hemorrhage secondary to anticoagulant therapy. *Am. J. Surg.*, 1960, *99*, 945–948.
4. Mendelsohn, S. N., and Iglauer, A. Acute abdominal condition due to bishydroxycoumarin (dicumarol) poisoning. *A.M.A. Arch. Int. Med.*, 1953, *92*, 760–762.
5. Pearson, S. C., and MacKenzie, R. J. Intestinal obstruction due to bishydroxycoumarin poisoning. *J.A.M.A.*, 1958, *167*, 455–456.
6. Spencer, R., Bateman, J. D., and Horn, P. L. Intramural hematoma of intestine, rare cause of intestinal obstruction: review of literature and report of case. *Surgery*, 1957, *41*, 794–804.

DUODENAL HEMATOMA INDUCED BY COUMARIN*

By JEROME F. WIOT, M.D., AARON S. WEINSTEIN, M.D.,† *and* BENJAMIN FELSON, M.D.

INTRAMURAL hematoma of the duodenum is an important and not uncommon entity which can be readily diagnosed by roentgenologic methods. Although described pathologically as early as 1894,[21] a detailed report of its roentgen aspects did not appear in the American literature until 1954, when Felson and Levin,[6] of this institution, described pathognomonic roentgen signs in 4 cases. Of the 23 proved cases found in the literature, 17 were the result of trauma.[2,3,6,7,11,15–17,21,22,25–29] In addition, 3 cases of spontaneous origin[3,18,19] and individual cases related to carcinoma of the pancreas,[19] pancreatitis,[15] and an obscure bleeding diathesis in an infant[9] have been recorded. Additional cases, similar pathologically, have also been noted as occurring elsewhere in the small bowel and in the colon.[12–14,18] Although intramural hematoma secondary to anticoagulant therapy has been described,[1,8,20] none of the reports mention roentgen contrast studies and none of the cases involved the duodenum.

Recently, we have encountered 2 patients with coumarin intoxication in whom roentgen changes in the duodenum resembling those previously observed in proved cases of intramural hematoma were seen. Since operation was not indicated in these cases, pathologic proof is lacking. However, in the light of the clinical and laboratory evidence and the subsequent spontaneous regression of the roentgen signs, the diagnosis can hardly be doubted.

REPORT OF CASES

CASE 1. E.L., a forty-one year old Negro male, was first admitted to the Cincinnati General Hospital on June 12, 1958 complaining of severe chest pain of three hours' duration. Physical examination was unremarkable except for a mild hypertension of 140/90. Laboratory studies, including serum amylase and a liver profile, were normal. An electrocardiogram revealed an acute posterior myocardial infarction. The patient was placed on a coronary occlusion regimen which included anticoagulants. The prothrombin time was maintained at about 20 per cent during hospitalization and on the twenty-eighth hospital day he was discharged on 5 mg. of coumarin per day.

Ten days after discharge he returned to the hospital complaining of cramping periumbilical pain, nausea, and vomiting of clear liquid for one week. On the day of admission the vomitus had become dark brown and the urine was grossly bloody. There was no history of trauma.

Physical examination on the second admission revealed a blood pressure of 120/100 and a pulse rate of 100. The temperature and respiratory rate were normal. He was in no acute distress. Positive findings were limited to the abdomen, which revealed generalized tenderness, most marked about the umbilicus.

The hematocrit was 33 per cent and the hemoglobin 10.6 gm. The white blood cell count was 11,000. The prothrombin time could not be determined, as a clot did not form. The bleeding time was seventy-five seconds and the clotting time was twenty-five minutes by the three tube method. Stool and vomitus were guaiac positive and the urine contained gross blood.

He was placed on an antacid, tincture of belladonna, and intravenous vitamin K. In approximately eight hours he stopped vomiting, and after thirty-six hours he was symptom-free. The prothrombin time gradually returned to normal during this period.

On the third day of the second hospital admission, an upper gastrointestinal series revealed marked changes in the mucosa of the entire duodenum distal to the bulb (Fig. 1, *A* and *B*). The mucosal folds appeared rigid and thick with the barium-filled troughs between them considerably narrowed. The normal feathery appearance was lost and the duodenal margins showed "spikes" of varying height. Longitudinal folds were not visible, so the mucosal

* From the Department of Radiology, Cincinnati General Hospital, and the College of Medicine, University of Cincinnati, Cincinnati, Ohio.

† Present address: Department of Radiology, Yale University School of Medicine, New Haven, Connecticut.



FIG. 1. Case I. July 24, 1958. (*A* and *B*) There are marked mucosal changes of the duodenum, with rigid thickened folds and narrow troughs between them. The pattern simulates a "picket-fence." The extrinsic pressure on the duodenum was more apparent on other roentgenograms.

pattern had a "picket-fence" appearance. There was a suggestion of localized extrinsic pressure on the medial wall of the descending limb of the duodenum. There was no evidence of obstruction. A follow-up study one week later showed considerable improvement in the duodenal pattern (Fig. 2) and after another week the gastrointestinal series was entirely normal (Fig. 3).

He was discharged on the nineteenth hospital day on 5 mg. coumarin per day and remained well except for one bout of epigastric pain, which subsided when he discontinued the medication for four days. He was last seen at the Cincinnati General Hospital in June, 1959, at which time he was asymptomatic.

CASE II. (Reported through the courtesy of Dr. James H. Scatliff, Yale University School of Medicine.) W.H., a sixty-seven year old white male, entered the Grace-New Haven

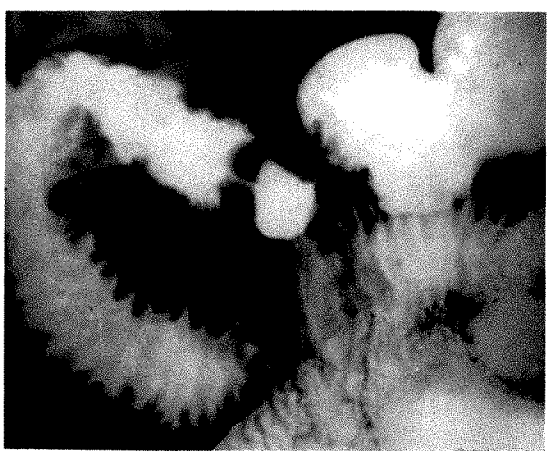


FIG. 2. Case I. July 31, 1958. Definite improvement has occurred, but the mucosal pattern is still recognizably abnormal.

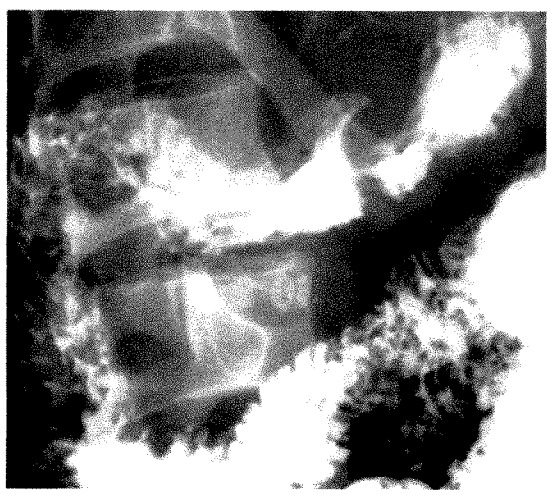


FIG. 3. Case I. August 8, 1958. The mucosal pattern is now entirely normal.

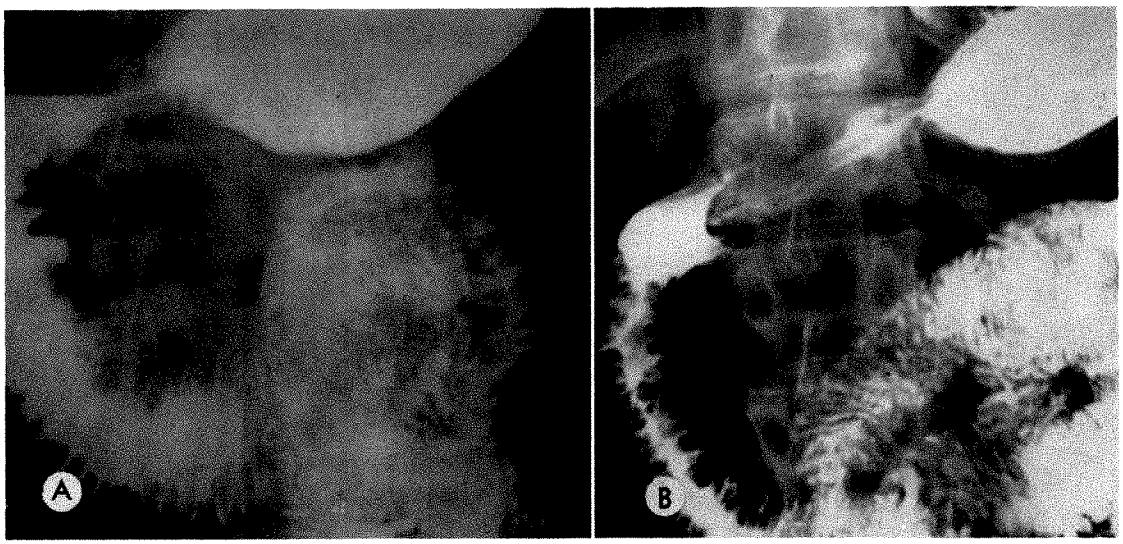


FIG. 4. Case II. December 1, 1959. (*A* and *B*) Abnormal mucosal pattern with "picket-fence" appearance, similar to the findings in Case 1. Extrinsic pressure upon the greater curvature of the distal end of the stomach is evident.

Community Hospital, November 24, 1959, with a history of bleeding into the skin and eye, pink urine, and black stools for about ten days. Lower abdominal pain had been present for several days. He had had an acute myocardial infarction in February, 1959, since which time he had been maintained on anticoagulants. Shortly before onset of the bleeding he had received overdoses of coumarin by mistake.

Physical examination revealed ecchymoses in the skin, conjunctival hemorrhage, and petechiae in the palate. His abdomen was slightly distended and tense, with mild tenderness of the right costovertebral angle.

The prothrombin time was greatly increased. The hematocrit was 29 per cent. Liver profile studies and serum amylase were normal. The electrocardiogram showed evidence of an old anterolateral myocardial infarction. A stool sample was reddish brown in color and guaiac positive. Anticoagulant therapy was discontinued and he was given two units of packed red blood cells, which resulted in prompt recovery.

An upper gastrointestinal series eight days after admission showed a striking swelling of the mucosal folds of the duodenum (Fig. 4, *A* and *B*). The troughs were narrowed and spiked. As in Case 1, longitudinal folds were lacking, resulting in a "picket-fence" pattern. The involved area was moderately distensible, and there was no obstruction. There was evidence of mild extrinsic pressure on the medial aspect of the descending duodenum and on the greater curvature of the distal end of the stomach. A barium enema examination was normal.

An upper gastrointestinal series thirty-eight days later showed a completely normal duodenal pattern with no evidence of extrinsic pressure on the duodenum or stomach (Fig. 5).

DISCUSSION

The roentgenographic changes of intramural hematoma of the duodenum are felt to be diagnostic when all of the manifestations are present. Classically, the lesion involves the descending and transverse segments of the duodenum but occasional cases have also involved the bulb and/or upper jejunum. The proximal portion of the abnormal segment shows only thickening of the mucosa. More distally, an intramural mass is present, associated with widening of the lumen and a coil-spring appearance due to crowding of the valvulae conniventes. Partial obstruction is common. Frequently, extrinsic pressure upon the greater curvature of the stomach and widening of the duodenal loop are seen, probably attributable to extraduodenal extension of the hematoma.

The findings in both of our cases differed somewhat from this description in that

there was no evidence of a localized intramural mass or obstruction, and a distinct coil-spring appearance was not apparent. Mucosal swelling and extrinsic pressure upon the stomach and duodenum were the only abnormalities. It is possible that the absence of an intramural mass is the result of failure of clot formation related to the coumarin therapy, or perhaps the diffuseness of the hematoma. The rapid clinical and roentgenologic recovery following medical treatment in our cases is consistent with the course of previously reported cases of unoperated intramural hematoma.

Felson and Levin[6] attempted to reproduce the roentgen findings of intramural hematoma of the duodenum experimentally by injecting water into the wall of a segment of normal small intestine removed at autopsy. They were able to duplicate the mucosal changes and intramural mass seen in clinical cases of intramural hematoma but not the coil-spring appearance (Fig. 6). The mucosal changes which they demonstrated experimentally very closely resemble the findings encountered in the present cases.

The association of hemorrhage with anticoagulant therapy is relatively frequent. In response to a questionnaire submitted to 228 physicians by Russek and Zohman[24] in 1953, 45.6 per cent reported one or more instances of serious hemorrhagic complications with 122 deaths from anticoagulant therapy for a variety of conditions. Thirty deaths from gastrointestinal hemorrhage were recorded, and this complication was second only to cerebral hemorrhage in frequency. Two-thirds of the deaths from gastrointestinal bleeding were attributed to pre-existing peptic ulcer but, in the remaining 10, the source of hemorrhage was not found.



Fig. 5. Case II. January 7, 1960. Normal duodenal pattern.

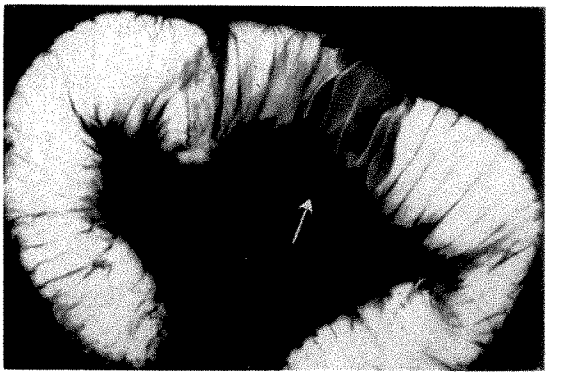


Fig. 6. Experimental attempt to duplicate the roentgen appearance of intramural hematoma by injection of water into the wall of a closed segment of jejunum filled with barium. The pattern resembles very closely the changes seen in Figures 1, *A* and *B*, and 4, *A* and *B*. (Reproduced with permission of *Radiology*.[6])

The mechanisms of hemorrhage in coumarin therapy are complicated. Briefly, these have been divided by Peyman[23] into four categories: (1) disturbance of normal blood clotting; (2) toxic effect on the capillary walls; (3) impairment of the normal adhesiveness of the platelets; and (4) accentuated recanalization of thrombosed arteries and veins. Not only is there great variation in the amount of the drug required to produce intoxication among different individuals but also in the same individual at different times.

The frequent occurrence of intramural hemorrhage in the duodenum as opposed to other areas of the bowel, in cases of trauma, is attributed to the fixation of this portion of the intestine at the ligament of Treitz with the trauma forcing the fixed retroperitoneal portion of the duodenum against the spine. The history of trauma,

however, must often be diligently sought, for in many reported cases this important fact was not uncovered until after the surgical findings.

As mentioned earlier, there have been 6 reported cases of intramural hematoma of the duodenum in which a history of trauma could not be obtained. In addition to the 2 cases secondary to coumarin, we have recently seen 3 other cases of duodenal hematoma in which trauma apparently did not play a role. The first of these was an elderly man with a very low prothrombin time secondary to alcoholic cirrhosis. The second was a nineteen year old boy with hemophilia. Both of these patients showed changes in the duodenum identical to those in the present report. The third patient, who also denied trauma, showed the classical roentgen findings described by Felson and Levin.[6] Thus, a total of 11 cases of duodenal hematoma in which a history of trauma could not be elicited have been documented. However, trauma as a factor in some of these cases cannot be excluded since, in the presence of a bleeding tendency, a minor unrecognized injury may have been sufficient to initiate the process. It is of interest that the last patient mentioned above and 1 of those reported by Felson and Levin were butchers. Possibly trauma to the abdomen occurred while they were carrying large slabs of meat, an everyday event in this occupation.

The reason that spontaneous hemorrhage so commonly occurs in the duodenal region of the gastrointestinal tract is obscure. However, there appears to be such a predilection, since a number of cases of diffuse duodenal hemorrhage have been reported in association with severe burns, tumor and trauma of the central nervous system, and major operative procedures. Recently, Katz[10] reported autopsy findings of "hemorrhagic duodenitis" in 25 cases of myocardial infarction. Although most of these patients were receiving anticoagulants immediately prior to death, none were apparently intoxicated by the drug. Roentgenograms were not available in any of the cases.

In the absence of roentgen evidence of an intramural mass or coil-spring appearance, the differential diagnosis of duodenal hematoma, as represented in the 2 cases in the present report, must include infarction secondary to vascular occlusion and such inflammatory conditions as pancreatitis and acute duodenitis. In these conditions the duodenal wall may be infiltrated by inflammatory exudate or hemorrhagic edema, resulting in a roentgen appearance similar to that caused by coumarin. The differential diagnosis is extremely important, as operation in the presence of an unrecognized bleeding tendency might have dire results. The history of coumarin administration, the low prothrombin time, the typical roentgen findings, and the response to therapy make the diagnosis of intramural hematoma caused by anticoagulants virtually certain.

SUMMARY

The roentgenologic appearance of intramural hemorrhage secondary to anticoagulant therapy has not been previously described despite the relatively frequent occurrence of gastrointestinal and other hemorrhagic complications of such therapy. The roentgen features differ somewhat from those of intramural hematoma caused by trauma, possibly because of its more diffuse nature and/or the absence of clot formation. Although the striking findings are also consistent with inflammatory changes in the duodenum, the history, laboratory, and roentgen findings readily establish the diagnosis.

Jerome F. Wiot, M.D.
Department of Radiology
Cincinnati General Hospital
Cincinnati, Ohio

We are indebted to Dr. James H. Scatliff, Yale University School of Medicine, for permission to report Case II.

REFERENCES

1. BERMAN, H., and MAINELLA, F. S. Toxic results of anticoagulant therapy. *New York J. Med.*, 1952, *52*, 725–727.
2. CAIRD, D. M., and ELLIS, H. Intramural haema-

toma of duodenum; report of case and review of literature. *Brit. J. Surg.*, 1958, *45*, 389–391.
3. COOLING, C. I. Subserous duodenal haematoma. *Brit. Med. J.*, 1953, *1*, 1051.
4. CULVER, G. J., and PIRSON, H. S. Intramural hematoma of duodenum; case report. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1959, *82*, 1032–1035.
5. DEY, D. L. Acute duodenal obstruction due to intramural haematoma. *M. J. Australia*, 1952, *1*, 708.
6. FELSON, B., and LEVIN, E. J. Intramural hematoma of duodenum; diagnostic roentgen sign. *Radiology*, 1954, *63*, 823–830.
7. GARFINKEL, B., WALESON, M., and FURST, N. J. Hematoma of duodenum; report of surgically proved case. *Am. J. Surg.*, 1958, *95*, 484–486.
8. GILBERT, A. E., and JORGENSON, N. C. Small bowel obstruction due to hemorrhage secondary to anticoagulant therapy. *Am. J. Surg.* 1960, *99*, 945–948.
9. GLASS, G. C. Hemorrhage in newly born infant, causing intestinal and biliary obstruction; report of case with necropsy. *Am. J. Dis. Child.*, 1937, *54*, 1052–1056.
10. KATZ, A. M. Hemorrhagic duodenitis in myocardial infarction. *Ann. Int. Med.*, 1959, *51*, 212–218.
11. KIRKPATRICK, W. E. Submucosal hematoma of duodenum; discussion and report of case. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 857–860.
12. KRATZER, G. L., and DIXON, C. F. Traumatic submucosal hematoma of midportion of ascending colon; report of case. *Proc. Staff Meet. Mayo Clin.*, 1951, *26*, 18–20.
13. LAMPERT, E. G., GOODFELLOW, J. G., and WACHOWSKI, T. J. Traumatic subserosal hemorrhage causing small bowel obstruction. *Ann. Surg.*, 1954, *140*, 768–770.
14. LIVERUD, K. Hematoma of jejunum with subileus. *Acta radiol.*, 1948, *30*, 163–168.
15. MCCLELLAND, J. R., THISTLETHWAITE, J. R., and GERWIG, W. H., JR. Obstruction of distal duodenum due to intramural hemorrhage. *Am. Surgeon*, 1958, *24*, 367–370.
16. MAGLADRY, G. W., JR., and MATHEWSON, G., JR. Duodenal obstruction due to trauma. *Stanford M. Bull.*, 1954, *12*, 205–206.
17. MELAMED, M., and PANTONE, A. M. Hematoma of duodenum; case report. *Radiology*, 1956, *66*, 874–876.
18. MOODY, E. E. Acute jejunal obstruction secondary to traumatic intramural hematoma. *Pediatrics*, 1957, *19*, 863–868.
19. OPPENHEIMER, G. D. Acute obstruction of duodenum due to submucous haematoma. *Ann. Surg.*, 1933, *98*, 192–196.
20. PEARSON, S. C., and MACKENZIE, R. J. Intestinal obstruction due to bishydroxy-coumarin poisoning. *J.A.M.A.*, 1958, *167*, 455–456.
21. PERRY, E. C., and SHAW, L. E. On diseases of duodenum. *Guy's Hospital Reports*, 1894, *50*, 171.
22. PESTER, G. H., and PEARTREE, P. Traumatic intramural hematoma of duodenum. *Am. J. Surg.*, 1958, *96*, 568–570.
23. PEYMAN, M. A. Significance of haemorrhage during treatment of patients with coumarin anticoagulants. *Acta med. scandinav.*, 1958, *162*, suppl. 339, 1–62.
24. RUSSEK, H. I., and ZOHMAN, B. L. Anticoagulant therapy in acute myocardial infarction; survey of specialists' opinions concerning indications, results and dangers. *Am. J. M. Sc.*, 1953, *225*, 8–13.
25. SCHREIBER, M. S. Reported by Dey.[5]
26. SNIDER, H. R. Acute duodenal obstruction secondary to intramural hematoma. *Surgery*, 1956, *39*, 860–864.
27. STIRK, D. I. Case of subserous duodenal haematoma. *Brit. M. J.*, 1953, *1*, 712.
28. SWAIMAN, K. F., ROOT, J. F., and RAILE, R. B. Coil-spring sign and intramural hematoma of proximal small intestine. *Am. J. Dis. Child.*, 1958, *95*, 413–416.
29. WATANABE, L. M., and INOUYE, M. R. Duodenal obstruction due to intramural hematoma. *California Med.*, 1956, *85*, 254–256.

NOTES ON A SMALL BOWEL EXAMINATION*

By JOSEPH L. MORTON, M.D.
INDIANAPOLIS, INDIANA

ELEVEN years ago Weintraub and Williams[8] described a method for a rapid small bowel examination using iced barium and saline solution. Since then, a similar method has been used by the author with very satisfactory results. Inquiry among my colleagues indicates that the technique is little used and is not well known. It is being described again because of its advantages, which are as follows: (1) areas of disease are easier to find; (2) the patient is examined in a single visit to the department, and in less time; and (3) by examining an area which frequently is bypassed, additional diagnoses are established.

TECHNIQUE

Barium of the suspended type (micropaque [R] is preferred), in the amount of 70 gm. in 200 ml. of ice cold tap water, is employed to examine the hypopharynx, esophagus, stomach and duodenum. The relative amounts are not important and can be varied to suit personal preference. On completion of the upper tract examination, note is made of the amount of barium remaining in the stomach, for in some patients the entire small bowel and colon will fill in thirty minutes. With practice it can be determined when these patients should be called back before the lapse of thirty minutes.

A glass of ice cold normal saline solution is then given every fifteen minutes until the examination is completed. The saline is made from tap water and table salt in the department (about 200 ml. per glass). It must be cold and should be isotonic for fastest transit time. Nauseated patients are placed on their right side on a hospital cart. Nausea is additionally controlled by inhalation of aromatic spirits of ammonia U.S.P. on a cotton pledget. It is well to explain to the patient that drinking the saline solution will shorten the examination and aid in elimination of the barium later. Experience with constipation after being examined with the older nonsuspended barium makes patients react more cooperatively to this procedure. We do not find it necessary to give the ambulatory nonconstipated patient a laxative with this technique.

Thirty minutes before completion of the stomach examination, the average patient is briefly fluoroscoped and a roentgenogram is made. Abnormalities requiring additional study are noted. Stasis, retroplacement of loops behind the spine and other changes are identified and disposition is made of the patient. If the barium has reached the cecum the examination is terminated; 31 per cent of the patients fall into this group. In an additional 34 per cent of patients, the barium is found in the terminal ileum. Reexamination of the latter group is made at fifteen to thirty minutes to study the ileocecal area. In 18 per cent the barium will have reached the midileum at thirty minutes and usually will have passed into the colon.

When passage of the barium is slower than this, the patient is evaluated at thirty minutes for the cause of delay.

Patients on a salt free diet because of nephritis, heart failure or other reason, should be given plain ice cold water. It is not as efficacious as the saline solution, but considerable motility still can be induced. Patients who have salt retention may gain 2–5 pounds if given the saline solution.

Occasionally more barium is required to study some questionable area, to relate various areas of compartmentation, or to confirm a serious lesion.

Patients are requested not to empty the bladder during the first thirty minutes or

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

until after they have been fluoroscoped. It is much easier to study the pelvic loops with the bladder filling the pelvis and elevating the small intestine.

ADVANTAGES

1. Areas of disease are easier to find. Stress demonstrates variations of pattern and motility and elicits pain in the abnormal small intestine. Polyps intussuscept with a slow forward motion and a quick plunger-like return which is very characteristic when once identified. Unsuspected cancers are revealed when barium backs up behind them (Fig. 1). Parasites such as *Ascaris lumbricoides* first show as negative defects and then usually ingest barium at forty-five minutes. Foreign bodies and enteroliths can be located for more detailed study. Peritoneal adhesions are indicated by increased deformation with rapid peristalsis.

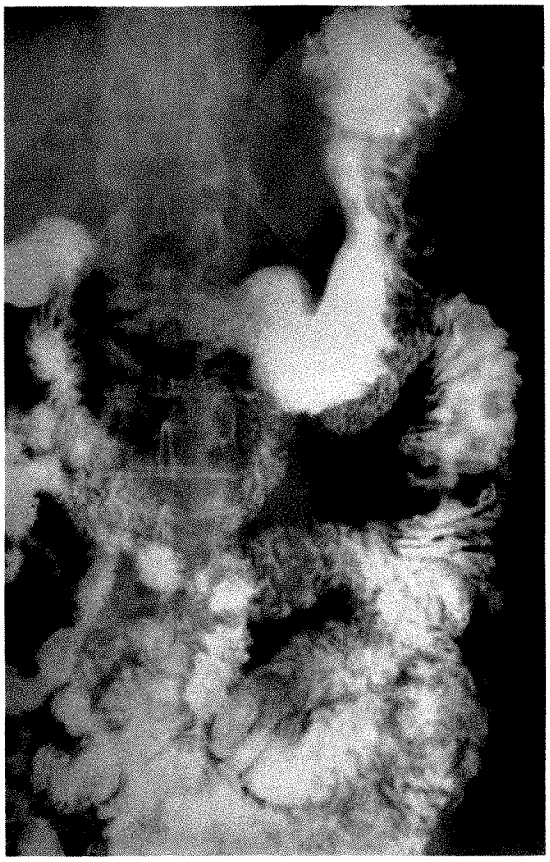


Fig. 1. Primary carcinoma of the jejunum in a seventy year old woman with blood loss anemia. Note stasis behind the lesion (arrow). Deep gastric folds were confusing initially.

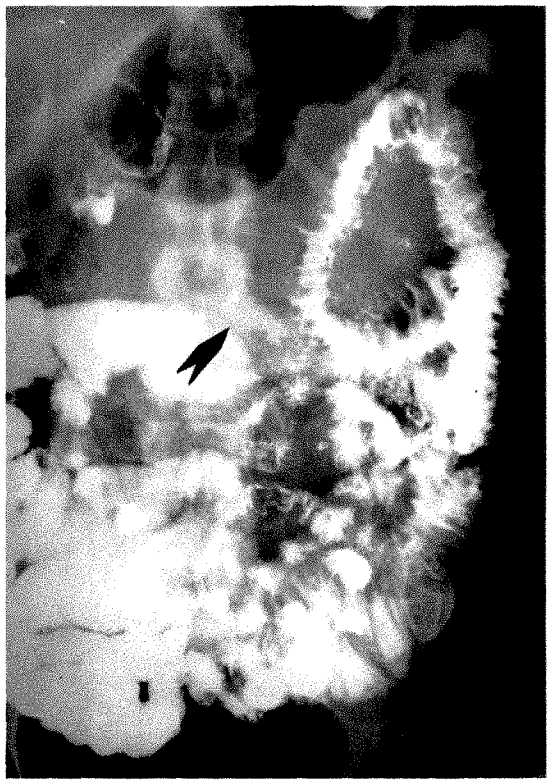


Fig. 2. Pancreatitis with mass (arrow), edema of loops and displacement of jejunum.

2. The method is convenient for the patient and the radiologist. Diagnoses are established and reported in a shorter time.

3. Unsuspected disease is discovered. Space occupying lesions are easier to evaluate when the small intestine is filled with barium (Fig. 2 and 3). Tumors of solid organs, aneurysms, and intraperitoneal and extraperitoneal lesions are outlined by the opacified intestine. We routinely combine excretory urography with a rapid small intestinal series for evaluation of paraaortic and iliac lymph node areas in lymphoma patients.

DISADVANTAGES

1. Ulcers are harder to see. When first using suspended barium, the radiologist is troubled by the necessity for more meticulous study of the mucosal pattern, as the craters are harder to identify. In our opinion, this is of little importance, for good



Fig. 3. Gas filled pocket (arrow), due to abscess following diverticulitis, visualized by filling small bowel loops.

spot roentgenography is always necessary in order to find many of the ulcers.

2. Narcotics will delay motility. If patients are seen within twenty-four hours after the administration of morphine, demerol or similar narcotics, they have protracted transit times. A decision must be made as to the probable value to the patient of the small intestinal study and, if the indications are not urgent, the examination should be terminated and resumed on another day.

FINDINGS AND COMMENTS

HERNIAS AND CONGENITAL CHANGES

The viscus occupying a hernial sac can be established and internal hernias recognized by this examination (Fig. 4). It is not rare to find femoral, inguinal or umbilical hernias which good clinical examiners have missed. Twice in infants we have observed umbilical hernias too small to be clinically identified. In the recognition of internal hernias, attention should be paid to agglutination of loops, recognized by the palpating hand, and to compartmentation (Fig. 5).

The retroplacement of loops behind the spine, seen first at the thirty minute recumbent study and confirmed by upright lateral roentgenograms, has been valuable in our experience.[5] This sign is present with paraduodenal hernias and adhesive bands with considerable regularity (Fig. 6). Admittedly, the number of such findings is not large, but the patients suffering from this compartmentation are likely to suffer for years unless there is a careful small bowel study.

Vestigial mesenteric attachments are particularly difficult to recognize without good roentgenograms. Only a slight straight-

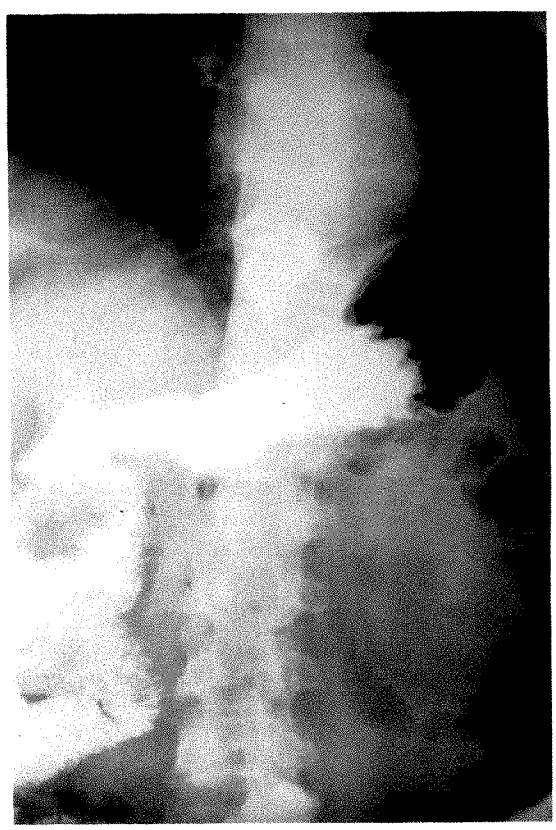


Fig. 4. Congenital failure of rotation with the jejunum displaced to the right and without a normal relationship to the superior mesenteric artery. The patient had been admitted twice in five years for recurrent obstruction.

ening or deflection of the loops may be observed. If the patient is emotionally stable and if the complaints are related to meals, it is worthwhile to repeat the examination when there is the merest suspicion of compartmentation with abnormal position of loops.

Multiple diverticula are of some clinical significance in macrocytic anemia. Reduplications and cysts can be demonstrated in relationship to bowel segments if all the loops are filled on one or two roentgenograms (Fig. 7).

INTRINSIC VASCULAR DISEASE

Although our clinical colleagues may require some persuasion, we find that ingestion of barium is no more dangerous than of food for cases of the acute mesenteric vascular accident. Recognized clinically by the acute onset, local mass and bleeding, the extent and improvement of the mesenteric occlusion can be demonstrated roentgenographically (Fig. 8 and 9). Judgement must be exercised in determining the best time for small bowel study, but knowledge of the behavior and size of the lesion improves its management. All cases do not require surgical intervention.

Obstructions of significance can be diagnosed from barium enema studies alone, and without oral barium. We have occasionally used hyperhydration of the patient with saline solution to prevent obstruction of the colon when, by accident or design, oral barium has been given.

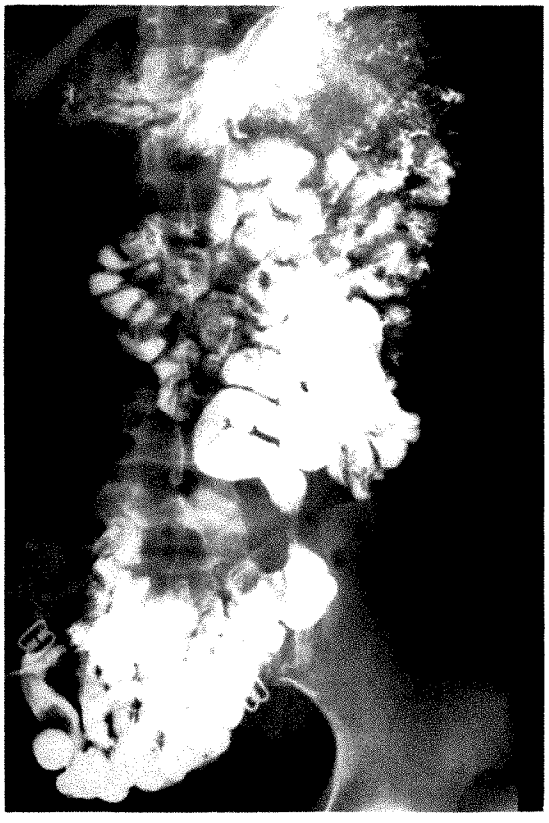


Fig. 5. Compartmentation by omental adhesion in the deep pelvis, presumed due to appendectomy in childhood. Recurrent vomiting, pain and bleeding forced surgical exploration after intermittent attacks over a three year period. The condition had been identified three years before surgery but the patient and surgeon initially were unwilling to accept the diagnosis.

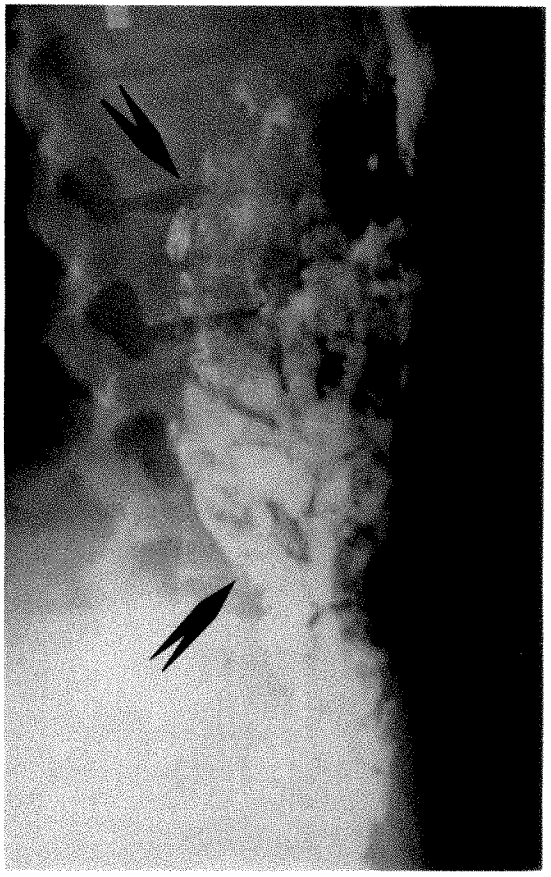


Fig. 6. Lateral upright view of the same case as Figure 4 showing loops (between arrows) behind the spine.

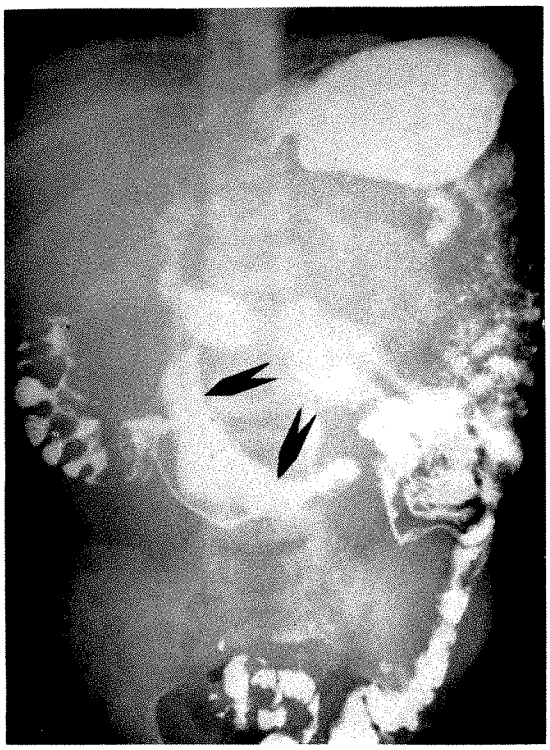


Fig. 7. Mesenteric cyst causing displacement and compression of the bowel and with the midabdominal loop of the small intestine (between arrows) too intimately bound to the mass to be preserved surgically. This is one of 3 such cases identified in the past six years, each of which had one midabdominal loop of the small intestine that required removal.

DISORDERED MOTILITY

Reference should be made to the work of Golden[3] who coined this useful term. Insofar as we can determine, in our examinations the same patterns are identifiable as those described by him, allowing for a condensation of the time internal. It seems more logical to stimulate the intestine to maximum effort than to fast the patient. Functional bowel states related to social maladjustment can be demonstrated to have irregular patterns of stasis, edema and hypermotile segments identical to those described by Friedman.[2] Depending on the interpretation, similar changes may be attributed to allergy, edema, nervous states or nutritional causes (Fig. 10). Generally, in malabsorption syndromes of the sprue type the barium moves slowly, so these cases can easily be distinguished from the other group. Some care should be taken to exclude electrolyte imbalance, as in heart, liver or kidney disease and anemia, as the cause for the delay. Insofar as we can determine, this differentiation is clinical and requires laboratory confirmation but it may be initially suggested by the gastrointestinal study.

PEPTIC ULCERS

A very interesting relationship exists between peptic ulceration, particularly with duodenal ulcers, and motility. Some patients show very rapid and others very slow transit times. At thirty minutes the barium had reached the rectum in almost twice as many ulcer patients as patients without demonstrable craters. No rate difference was found between those having only deformity of the cap, without demon-



Fig. 8. Acute vascular occlusion (arrow) in the terminal ileum. This was treated conservatively with complete return of the pattern to normal after a clinical diagnosis.

strable ulcers, and those without any evidence of ulcers in this respect. Of 514 ulcer patients having demonstrable craters, the barium had reached the rectum in 3.1 per cent, whereas, of the remaining 1,236 patients, it had reached the rectum in only 1.8 per cent. This is not a new observation, according to Alvarez.[1] The slow transit times are complicated problems related to perforation, inflammation and duodenal ileus. In 10.7 per cent of the ulcer group, barium did not pass the jejunum at thirty minutes as compared to 2.3 per cent of the non-ulcer group.

An impression is gained from the pattern of repeat examinations that patients with average transit times are easier to control and do not return for repeat examination when released from medical management. Sometimes a patient with an initially slow transit time becomes hypermotile as the ulcer heals. Others revert from hypermotility to average transit times with healing of the ulcer. The patient without evidence of hypermotility appears unlikely to have a serious duodenal ulcer.

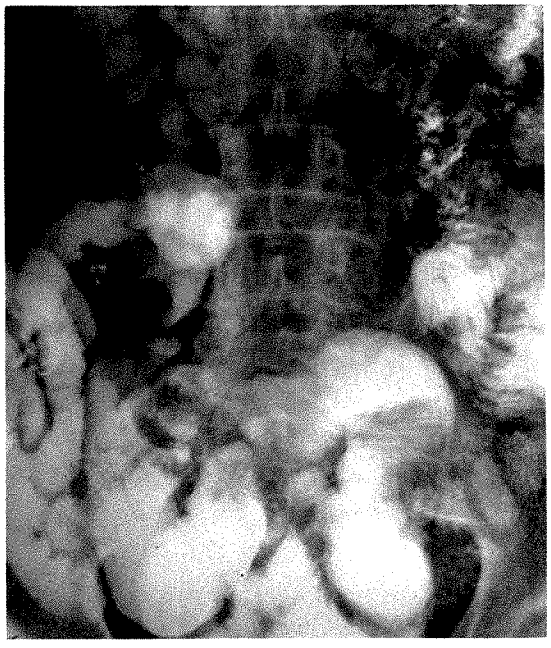


FIG. 9. Acute vascular occlusion (arrow) of undetermined cause. Exploratory laparotomy six months after this study showed residual adhesion between two loops; resection was not necessary.

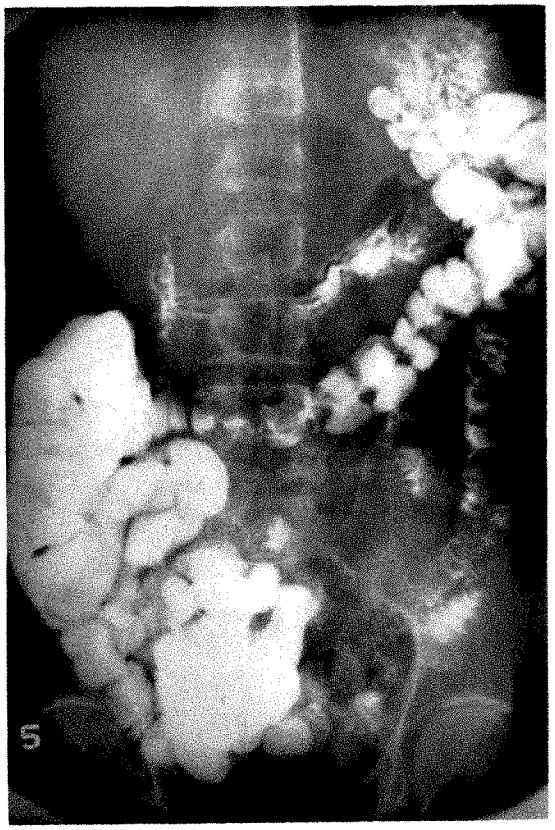


FIG. 10. Illustration of the rapid transit of barium to the sigmoid colon (fifteen minutes) in an adolescent girl; the cause was not recognizable clinically.

Attention was strongly called to this hypermotility relationship by fluoroscopy of the first patient with a recognized Zollinger-Ellison syndrome. She had had diarrhea for many years with copious stools several times a day before the acute ulcer pain developed. A similar patient has been described with watery diarrhea and islet cell tumor without peptic ulcer.[7] Parenthetically, note should be made of the difficulty in adequately studying the jejunum in such cases. The continuous voluminous gastric hypersecretion dilutes the barium rapidly, making a satisfactory mucosal pattern study very difficult, if not impossible. The patulous, slightly obstructed atonic cap and second portion of the duodenum add to the problem. Unless extreme care is taken, the jejunal ulcer will be hidden by the stomach and adjacent jejunal loops. Changes in caliber of the lumen and

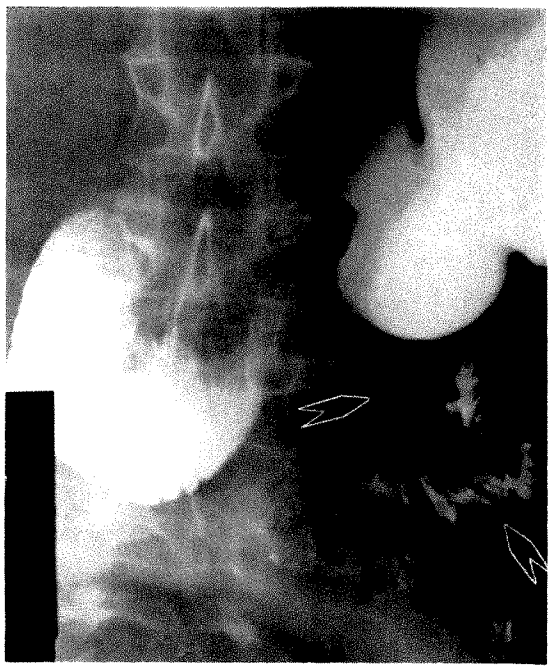


FIG. 11. This patient had a cast syndrome with a high degree of small intestinal obstruction at the duodenojejunal junction (arrows). It was postulated that marked edema was present from the appearance of the few jejunal loops seen at forty-five minutes. Edema was thought to be the cause of obstruction and a positive indication for surgery, which confirmed the roentgen findings. Rapid and complete recovery occurred.

sharp kinks or knuckles of small intestine should be especially watched when typical gastrosuccorrhea and duodenal ileus are encountered.

We have speculated that the rapid gastric emptying rate and the hypermotile small intestine are reverse facets of the hypersecretion factor in ulcer genesis; *i.e.*, peptic and acid secretions are induced by the transit of food through the small intestine without sufficient food remaining in the stomach to protect the lining and to neutralize the enzymes and acid formed. In other words, peptic ulcers occur either in patients with hypersecretion or in those who empty their stomach too soon to neutralize the normal secretions. If this be true, routine measurement of motility would provide useful clinical information with indications for either anticholinergic drugs or antacids in the management of ulcer patients.

Other conditions associated with hypermotility in our material are occasional cases of hyperthyroidism, emotional instability, gallstones and nonspecific acute enteritis.

INFECTION

The constitutional reaction to infection has many interesting relationships. Some patients with duodenal ileus from the superior mesenteric artery syndrome appear to have had recent changes in nutrition or in protein or electrolyte balance related to infections immediately preceding the development of acute symptoms. A seventeen year old boy, in a body cast, was rapidly deteriorating without much fever but with leukocytosis, vomiting and electrolyte imbalance. His original injury was a fracture of the femur four months before, treated by an intramedullary nail. Osteo-

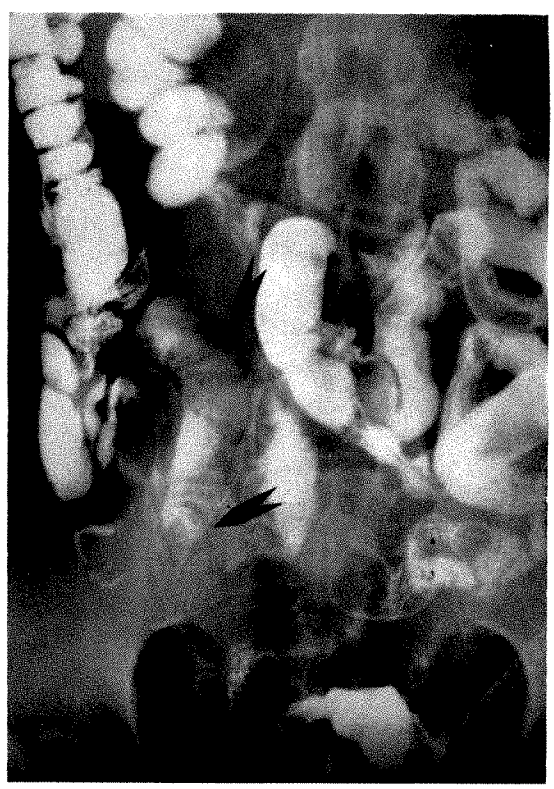


FIG. 12. Persistent distention and compression of the terminal ileum (arrows) due to Jackson's membrane found at surgery.

myelitis developed and, on admission here, the pin was removed and a body cast applied. A duodenal ileus followed. The interesting feature is, however, that the distal edematous loops were recognized on the roentgenogram and verified at surgery (Fig. 11). After duodenojejunostomy, the postoperative course was smooth.

Less dramatic episodes are sometimes seen with the gradual development of duodenal ileus after an infection or diarrhea which has been treated with antibiotics. We have found such a patient to have normal duodenal motility prior to such an infection, only to discover, a few months later, a typical "superior mesenteric artery syndrome." At times the unfortunate patient with chronic peritonitis and adhesions seems to undergo similar obstructive episodes after infections. An old established regional ileitis similarly may be adversely affected by respiratory infections. Such episodes of recurrent inflammation have been noted by Parsons,[5] who attributes them to edema from adhesions, with eventual obstruction.

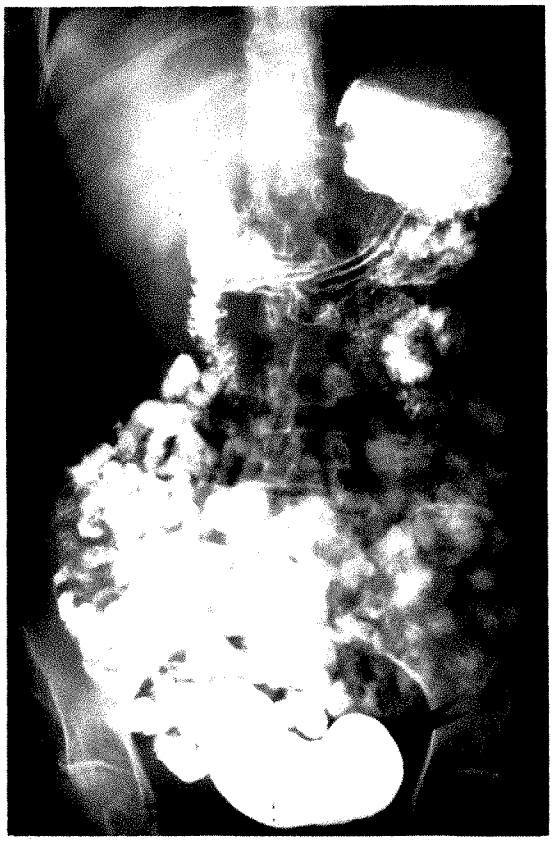


Fig. 13. Distended small bowel loop of the ileum (arrows) in a patient who had had colicky complaints throughout her life. Patient was of small stature and slight habitus. Operation was refused. This loop was seen to be rotated medially and laterally and to be inverted in the midabdomen on each of four examinations over a five year period.



Fig. 14. Subacute small bowel obstruction (arrows) from intestinal adhesions and pelvic peritonitis. Since the patient tolerated food, barium was given to identify the obstruction and the amount of disturbance of the intestinal tract. A single band at the terminal ileum was found at surgery.

CECAL AREA LESIONS

We find with Golden,[3,4] that the cecal area can profitably be studied from above for such diverse conditions as Jackson's membrane (Fig. 12), congenital bands and webs, short patches of obstruction from many causes (Fig. 13 and 14), ileitis (Fig. 15 and 16), appendicitis (Fig. 17), abscess, granulomas and tumors. Extrinsic tumors of the appendix and cecum and enlarged iliac lymph nodes can protrude into the barium column to give graphic evidence of their existence (Fig. 18). One case of intussusception of the ileal cecal valve through a tight submucosal constriction was found and later relieved by surgery. Another case of ileal intussusception in an eleven year

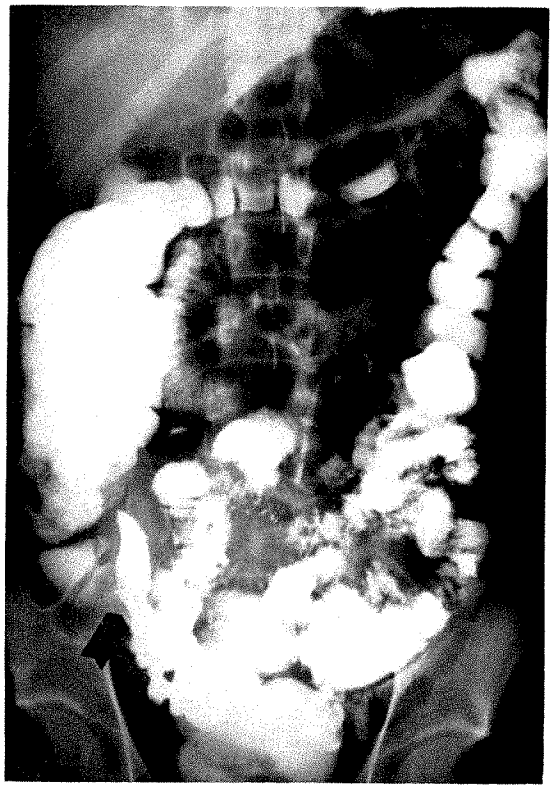
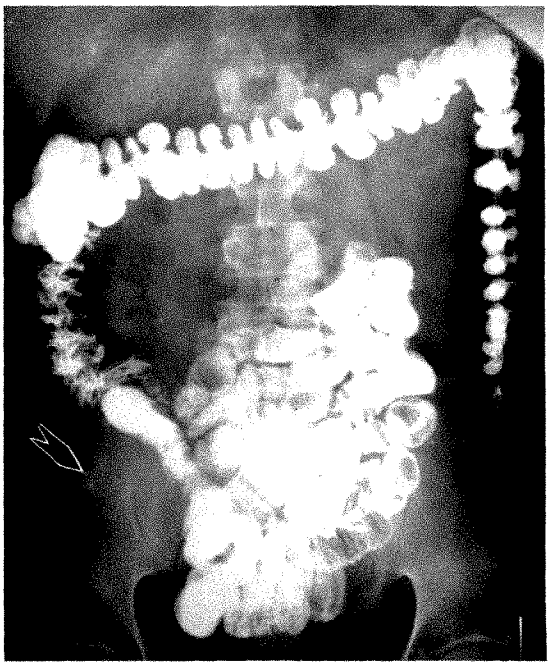


FIG. 15. Early thickening and edema of the terminal ileum (arrow), thought to be due to regional ileitis in the developing stage. This is a not uncommon finding that requires a small bowel examination for verification as it is a diagnosis of exclusion, since only this minor change is present.

old boy with healed duodenal ulceration was almost accidentally found at the small bowel study when there had been no prior clinical or roentgenologic suspicion. No polyp or diverticulum to explain the intussusception was found at surgery and he has been well since then. We suppose this to





top right

FIG. 16. Advanced stage of regional ileitis (between arrows) with cecal involvement.

bottom right

FIG. 17. An organized appendiceal mass of five years' duration (arrow) is outlined by small bowel loops. The diagnosis, not established by previous examinations made elsewhere of the gastrointestinal tract, was verified surgically.

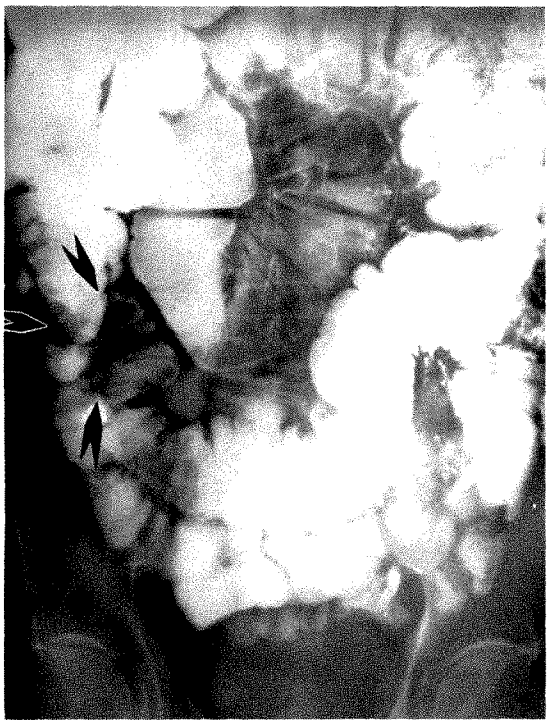


Fig. 18. Carcinoma of the colon with extraluminal mass (arrows) slightly obstructing the small bowel. The obstruction had not been clinically identified. The barium enema study was not diagnostic but this appearance of small bowel obstruction and the demonstration of the mass between the cecum and ileum by compression studies established the need for surgery.

be another example of hypermotility with duodenal ulcer, for we are certain that an ulcer was present a year earlier.

None of these cases are common enough to lead to directed examinations by the clinician but taken together they make up a respectable number of serious diseases discovered by routine examination.

SUMMARY

A technique for the physiologic forcing of barium through the small intestine at a rapid rate is again presented in the hope that, because of its simplicity and usefulness, it will be more widely adopted. The entire small bowel can be visualized in one hour in the normal patient with, at most, two roentgenograms. The discovery of unexpected disease and guides for therapy are enough to justify the routine use of the method. The adoption of adequate gonadal protection, rapid screening, fast screens and film, high kilovoltage and modern developers more than compensates for the possible additional exposure and makes radiation dosage tolerable. The time saved should alone justify the additional expense of suspended barium, without considering the scientific reasons for its use.

Department of Radiology
St. Vincent's Hospital
120 West Fall Creek Parkway
Indianapolis, Indiana

The author wishes to express thanks to the entire Radiology Department staff, and especially to Miss Phillis Willoughby of St. Vincent's Hospital, for aid given in preparing this paper.

REFERENCES

1. Alvarez, W. C. An Introduction to Gastroenterology. Fourth edition. Paul B. Hoeber, Inc., New York, 1948, p. 123.
2. Friedman, J. Roentgen studies of effects on small bowel from emotional disturbances. *Bull. Univ. Minnesota Hosp.*, 1953, *24*, 593–602.
3. Golden, R. Abnormalities of small intestine in nutritional disturbances: some observations on their physiologic basis (Carman lecture). *Radiology*, 1941, *36*, 262–286.
4. Golden, R. Radiologic Examination of the Small Intestine. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1959, p. 13.
5. Parsons, P. B. Paraduodenal hernias. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *69*, 563–589.
6. Scudamore, H. H., Hagedorn, A. B., Wollaeger, E. E., and Owen, C. A., Jr. Diverticulosis of small intestine and macrocytic anemia with radioactive vitamin B12 absorption studies. *Proc. Staff Meet. Mayo Clin.*, 1958, *33*, 651–657.
7. Verner, J. V., and Morrison, A. B. Islet cell tumor and syndrome of refractory watery diarrhea and hypokalemia. *Am. J. Med.*, 1958, *25*, 374–380.
8. Weintraub, S., and Williams, R. G. Rapid method of roentgenologic examination of small intestine; preliminary report. Am. J. Roentgenol. & Rad. Therapy, 1949, *61*, 45–55.

MUCORMYCOSIS OF THE COLON

By EDWARD DE FEO, M.D.
SCHENECTADY, NEW YORK

MEMBERS of the *Mucoraceae* family of fungi are common saprophytes and laboratory contaminants. They have been recognized as pathogens in man, on occasion, since the last century.[1,2] Prior to 1954, accounts have been rather rare; since that time, however, articles of infection in man have been numerous. Most of these have been concerned with involvement of the paranasal sinuses, central nervous system, or the lungs. Less common have been reports of cases involving the intestinal tract. Within the past three years, 4 cases have been discovered at this institution which will be reported elsewhere. One of these, in which the colon is involved, is reported below.

REPORT OF A CASE

A fifty-one year old white male was admitted to Ellis Hospital in October, 1956, three months following circumcision for an ulceration of the prepuce of the penis. At admission he complained of early fatigue and slight epigastric "burning" sensation. On physical examination there were present bilaterally-enlarged inguinal lymph nodes, small axillary lymph nodes, a healed furuncle in the right axilla, and moderate spleen enlargement.

Initial blood studies revealed: red blood cells 2.4 million; hemoglobin 58 per cent, 9 gm.; packed cell volume 28 per cent; platelets 78,000; white blood cells 2,000 with stabs 2 per cent, segm. 16 per cent and lymphocytes 82 per cent; slight anisocytosis; and immature and atypical lymphocytes. The bone marrow study demonstrated chiefly primitive cells having the characteristics of lymphoblasts and an increase in mature lymphocytes. Erythroid tissue was diminished but adequate. Rare megakaryocytes were seen. On review of the prepuce section, the corium was noted to be infiltrated by lymphoid and plasma cells, few eosinophils, and many large mononuclear cells in forms of distinct nodules or gathered about blood vessels and nerves. The impression of both was lymphoblastic lymphoma.

The patient was given one transfusion and treated with ACTH, 20 units by intravenous drip daily for ten days, and then 30 mg. of meticorten. Within a few days his white blood cell count dropped and remained between 400–800 cells. Radiation therapy was given to the splenic area with a surface absorbed dose of 1,427 rads. Roentgenograms of the spine, chest and intestinal tract were negative except for a few small diverticula in the descending colon (Fig. 1).

The patient was discharged after three weeks and maintained on meticorten for seven months. During this period he received several units of packed cells, testosterone and "spray" radiation of 105 r exposure dosage on two occasions.

He was re-admitted in June, 1957 with complaints of increasing fatigue, weakness, chills, fever, abdominal cramps, left lower abdominal pain, and pain at the sites of upper incisor

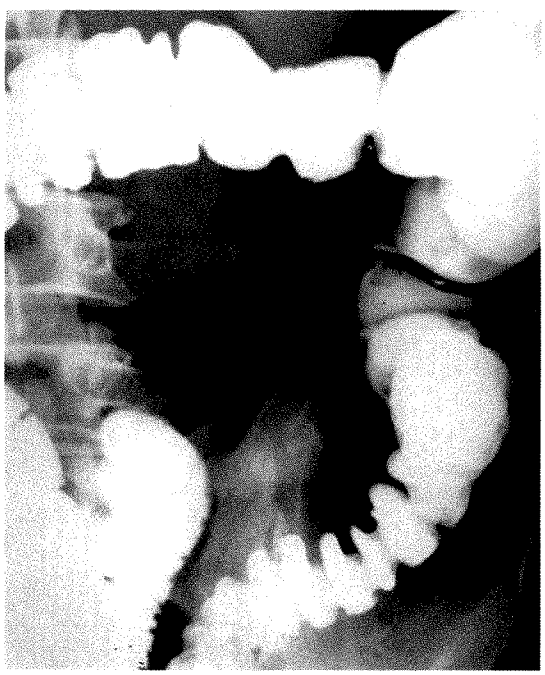


FIG. 1. Barium enema examination in October, 1956 was negative except for several small diverticula of the descending colon just above the iliac crest level.

periapical infections and cellulitis. He was tender in the abdominal left lower quadrant but no spasm or rigidity was noted.

The teeth were extracted and the abscesses were drained without complication. Abscess culture and blood cultures were sterile. Large amounts of penicillin, streptomycin and chloromycetin were administered. A barium enema study (Fig. 2 and 3) revealed an annular defect of the descending colon outline where diverticula were noted previously. There was a short zone of involvement and abrupt transition to a normal outline. A few diverticula were seen in the area. The bowel was not fixed in position during fluoroscopy. Because of the history, lymphomatosis involvement was considered and an estimated dose of 1,500 rads was delivered to this zone. He was discharged in ten days at which time there was no temperature elevation.

Final admission followed in one week because of no bowel movements and increasing severity of abdominal pain which was aggravated by movement. He had chills and a spiking fever up to 105° F. The abdomen was distended, tympanitic, and there was a palpable mass in the lower left quadrant. At this time, his red blood cell count was 2 million, hemoglobin 6.7 gm., platelets 70,000 and the white blood cell count 150. A roentgenogram of the abdomen showed intestinal obstruction at the descending colon. Penicillin, chloromycetin, streptomycin and hydrocortisone therapy was continued.

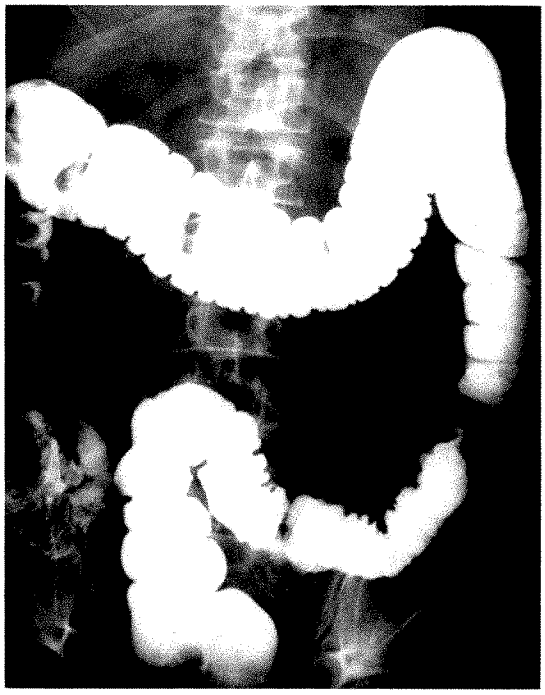


Fig. 2. Barium enema examination in June, 1957, about two weeks prior to laparotomy, revealed a short, irregular, annular narrowing of the colon at the same level. Two diverticula were noted on the lateral aspect of the colon at the margin of the lesion.

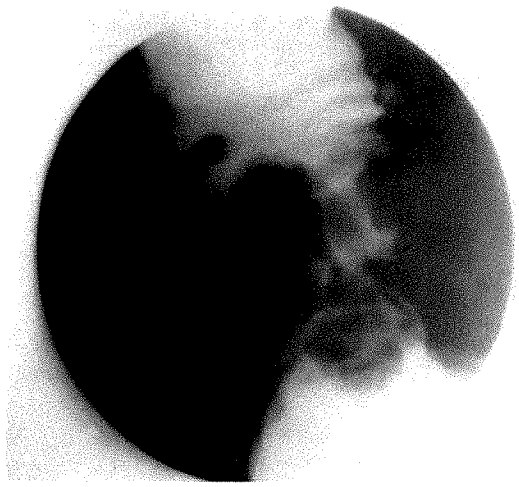


Fig. 3. Enlargement of involved portion of Figure 2.

Although he was considered a poor surgical risk, after three days a laparotomy was performed. Foul-smelling material was encountered and portions of necrotic descending colon wall were recognized. The area was drained and the abdomen closed partially. The patient became very weak and expired on the third day.

At postmortem examination 300 cc. of clear amber fluid was removed from the peritoneal cavity. There was thickening and marked inflammation of one short area of the descending colon. In dissecting the colon in this zone, a mass of fecal material was found in the mesocolon. The peritoneum at the site of surgery and around the colon was inflamed and showed yellowish fibrin pus deposits. On microscopic section (Fig. 4) there were necrosis and leukocytic infiltration, extensive submucosal hemorrhage and thrombosed vessels. The thrombi were formed by a meshwork of hyphae and red blood cells. Hyphae, in masses, were growing through the vessel walls into adjacent tissue which was infarcted. These had the typically wide diameter of mucormycosis, were



FIG. 4. Photomicrograph of section showing hyphae growing into the colon wall.

nonseptate and coenocytic, with branching almost at right angles. No cultures were made.

A secondary branch of the pulmonary artery was occluded by a thrombus formed by masses of the same type of fungus. These had grown into adjacent lung tissue where nearby were foci of bronchopneumonia.

There were a few superficial denuded areas in the mucosa of the stomach, but no fungi were found at those sites. The spleen had two small infarcted areas. The sinusoids were wide and focal interstitial hemorrhage was present. Normal bone marrow cells were replaced almost completely by atypical immature large cells with large vacuolated nuclei containing chromatin resembling lymphoblastic cells. Only a few red blood cells were present. The polychromatophilic cells of the granulocytic series were absent almost completely. The diagnosis was lymphoblastic leukemia.

No fungus invasion of any organs other than the colon and lung was found.

DISCUSSION

Mucoraceae hyphae are wide, with diameters ranging from 4–20 μ, nonseptate and having numerous branches, often extending outward at right angles.[10] Genera cannot be identified except by culture and usually this is not done prior to tissue fixation, since the presence of the fungus is not often suspected. Where cultures have been made, a member of the genus *Rhizopus* was usually found to be the responsible organism.[8,11]

Spores of the organisms are air borne and are believed to enter the body by either inhalation or by way of the intestinal tract, although spores are not found in the body. There is a strong tendency of the fungi to invade blood vessels and produce intense, localized inflammatory reaction, necrosis and thrombosis. The favored route of spread is along blood vessels or lymphatics, especially, in the perineural zones. In some instances only one system is involved, but, in almost as many, the disease is disseminated.

In a great majority there is an associated chronic, debilitating disease, usually leukemia or diabetes, and also, less commonly, nephritis, tuberculosis, liver abscess and diarrhea.[8,16] Several case histories indicate no such association.[5,14] The disease has been produced experimentally in animals in the acute toxic phase of alloxan diabetes.[4,9] The sudden increase in the number of reported cases has coincided with the

use of large amounts of antibiotics and cortisone, and the latter, especially, appears to be a significant factor affecting both defense mechanism and fungus spread and proliferation.[3,9]

Because of the production of thrombi in significant vessels by hyphae masses, the survival time of those patients treated to prevent bacterial infection may be less than of those succumbing to bacteria. The reports of deaths indicate only a very short period of survival from the time of onset of the disease.

Five cases with involvement of the intestine have been collected by Baker *et al.*[2] in addition to reported single cases.[6,7,13,17] The palate or esophagus was involved once. The stomach was involved in 2 patients, the small intestine in 3 and the colon in 4. In 1 case the disease was disseminated. The appearance of the lesion of the cecum in Clark's[6] diabetic-leukemic patient was described, following a gastrointestinal series, as consistent with a carcinoma or leukemic infiltration of adjacent lymph nodes.

When pathologic findings are discussed, the presence of intense inflammatory reaction with bowel wall edema, exudate and entrapped leukocytes is described. There is a sharp demarcation between involved and normal intestine.[15] Necrosis or ulcerations always are present with thrombosis in adjacent vessels. Hyphae are found growing from bowel wall into submucosal vessels and in the thrombi.

The sequence of events in this reported case cannot be established, however, with the short zone of involvement; with the presence of adjacent uninvolved diverticula, it may be assumed that perforation due to thrombosis or hyphae penetration was the initial event rather than perforation of a diverticulum.

This case emphasizes the error of irradiating a lesion without biopsy after large amounts of cortisone have been given, when there is a marked depression of the white blood cell count and a diagnosis seemingly has been established.

In similar cases with bowel defects and suspicion of perforation, prompt surgery may be indicated rather than permitting more infarction and spillage during a valueless delay. With the increasing use of massive therapy in diabetic or leukemic patients, more such cases are predicted.

SUMMARY

1. A case is reported of a patient with the diagnosis of lymphatic leukemia receiving large doses of ACTH and antibiotics who developed an annular lesion of necrosis and perforation of the descending colon due to mucormycosis.

2. Since the use of such therapy is increasing, a marked rise in the incidence of mucormycosis is predicted in patients with diabetes, leukemia and in those chronically ill, possibly with similiar types of intestinal defects.

Ellis Hospital
Schenectady 8, New York

REFERENCES

1. BAKER, R. D. Mucormycosis—new disease? *J.A.M.A.*, 1957, *163*, 805–808.
2. BAKER, R. D., BASSERT, D. E., and FERRINGTON, E. Mucormycosis of digestive tract. *A M.A. Arch Path.*, 1957, *63*, 176–182.
3. BAKER, R. D., SCHOFIELD, R. A., ELDER, T. D., and SPOTO, A. P., JR. Alloxan diabetes and cortisone as modifying factors in experimental mucormycosis (Rhizopus infection). *Fed. Proc.*, 1956, *15*, 506–507.
4. BAUER, H., FLANAGAN, J. F., and SHELDON, W. H. Effects of metabolic alterations on experimental Rhizopus oryzae (mucormycosis) infection. *Yale J. Biol. & Med.*, 1956, *29*, 23–32.
5. BLANKENBERG, H. W., and VERHOEFF, D. Mucormycosis of lung; case without significant predisposing factor. *Am. Rev. Tuberc.*, 1959, *79*, 357–361.
6. CLARK, R. M. Case of mucormycosis of duodenum, liver and cecum. *Gastroenterology*, 1957, *33*, 985–990.
7. CRAIG, J. M., and FARBER, S. Development of disseminated visceral mycosis during therapy for acute leukemia. *Am. J. Path.*, 1953, *29*, 601.
8. DWYER, G. K., and CHANGUS, G. W. Rhinomucormycosis resulting in fatal cerebral mucormycosis. *A.M.A. Arch. Otolaryng.*, 1958, *67*, 619–623.
9. ELDER, T. D., and BAKER, R. D. Pulmonary

mucormycosis in rabbits with alloxan diabetes; increased invasiveness of fungus during acute toxic phase of diabetes. *A.M.A. Arch. Path.*, 1956, *61*, 159–168.

10. Feinberg, R., and Risley, T. S. Mucormycosis infection of arteriosclerotic thrombus of abdominal aorta; report of case. *New England J. Med.*, 1959, *260*, 626–629.
11. Gabriele, O. F., Mucormycosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *83*, 227–235.
12. Gregory, J. E., Golden, A., and Haymaker, W. Mucormycosis of central nervous system; report of 3 cases. *Bull. John Hopkins Hosp.*, 1943, *73*, 405–419.
13. Harris, J. S. Mucormycosis; report of case. *Pediatrics*, 1955, *16*, 857–867.
14. Lloyd, J. B., Sexton, L. I., and Hertig, A. T. Pulmonary mucormycosis complicating pregnancy. *Am. J. Obst. & Gynec.*, 1949, *58*, 548–552.
15. Moore, M., Anderson, W. A. D., and Everett, H. H. Mucormycosis of large bowel. *Am. J. Path.*, 1949, *25*, 559–567.
16. Stefanini, M., and Allegra, S. Pulmonary mucormycosis in acute histiocytic leukemia. *New England J. Med.*, 1957, *256*, 1026–1030.
17. Watson, K. C. Gastric perforation due to fungus mucor in child with kwashiorkor. *South African M. J.*, 1957, *31*, 99–101.

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THE ROENTGENOLOGIC FEATURES OF CARCINOMA IN CHRONIC ULCERATIVE COLITIS*

By JOHN R. HODGSON, M.D., *Section of Roentgenology, and*
WILLIAM G. SAUER, M.D., *Section of Medicine*
Mayo Clinic and Mayo Foundation†
ROCHESTER, MINNESOTA

THE occurrence of carcinoma in chronic ulcerative colitis is a well-established fact; numerous authors have called attention to this relationship.[1–3] Less well known are the roentgenologic features of the carcinoma associated with ulcerative colitis. In order to clarify the roentgenologic manifestations of this disease and to explore the factors that may permit roentgenologic differentiation between carcinoma and stricture or polypoid hyperplasia in chronic ulcerative colitis, 49 cases of roentgenologically suspected carcinoma associated with ulcerative colitis were reviewed. Of the 49 patients, 27 were men and 22 were women.

A total of 29 of the original 49 patients were ultimately shown to have carcinoma of the colon superimposed on chronic ulcerative colitis. In all 29 cases the diagnosis was proved by pathologic examination. Among the other 20 cases in which there was roentgenologic suspicion of a malignant lesion, 2 were cases of carcinoma of the colon in which there was no evidence of ulcerative colitis; in 7 others an operation was not performed because other clinical findings did not corroborate the roentgenologic diagnosis; in another 7 cases resection of the colon was performed for chronic ulcerative colitis with what proved to be stricture or polypoid hyperplasia; and in the remaining 4 cases exploration revealed ulcerative colitis but failed to reveal carcimona, and no resection was performed.

In 1949, Sauer and Bargen[4] in a report of 41 cases of carcinoma complicating chronic ulcerative colitis stated that the distribution of carcinomas was as follows: 19 in the rectum, 5 in the sigmoid, 4 in the descending colon, 6 in the splenic flexure, 6 in the transverse colon, 3 in the hepatic flexure, 4 in the ascending colon, and 10 in the cecum. (More than one carcinoma was present in some patients.)

The present review of the roentgenologic manifestations of carcinoma in ulcerative colitis will be concerned only with neoplasms occurring above the level of the rectosigmoid, since lesions in the rectum may be more easily and accurately demonstrated by proctoscopy. The distribution of carcinoma was as follows: transverse colon, 14; splenic flexure, 4; hepatic flexure, 2; descending colon, 7; ascending colon, 6; cecum, 3; sigmoid, 1. Of interest is the relatively large number of occurrences in the transverse colon as well as the solitary case in the sigmoid. Multiple lesions were present in some cases. In all but 2 of the 29 cases the entire colon was involved with ulcerative colitis. When the carcinoma occurred in segmental ulcerative colitis, as it did in the remaining 2 cases, it showed no features different from those of the carcinoma in the other 27 cases.

CLINICAL OBSERVATIONS

It was pointed out in a previous article by one of us[4] that chronic ulcerative colitis began in early adult life in the majority of patients in whom associated carcinoma developed. Few patients were more than thirty-five years of age when symptoms began. In most instances these patients tended to do fairly well on treatment for a time, but they usually had rather severe

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.
† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

exacerbations during the course of their illness.

Most patients noted a change in the character of their symptoms which tended to point to the fact that a complicating malignant lesion was developing or was present. All had had ulcerative colitis a number of years before the change in the character of the symptoms occurred. The outstanding new symptoms consisted of progressive loss of weight, abdominal pains or cramps differing from the distress usually associated with colitis, an increase in the number of stools, and an increase in the appearance of blood in the stools. In some instances an abdominal mass was noted by the patient. Practically all patients complained of weakness. Some presented signs and symptoms of intestinal obstruction. Hypochromic anemia of rather severe degree was a fairly constant finding in association with the malignant change.

In the present group of 29 cases the average age at the time of discovery of the carcinoma was thirty-five years; the youngest patient was twenty-eight, the oldest sixty-two. The number of years that chronic ulcerative colitis had been present averaged seventeen. The shortest history of the disease was one year and the longest thirty-nine years.

ROENTGENOLOGIC FEATURES

The form carcinoma may take in chronic idiopathic ulcerative colitis may vary considerably. In general, five morphologic groups may be recognized roentgenologically: (1) constricting, (2) annular, (3) sessile, (4) polypoid, and (5) scirrhous. The term "scirrhous" actually does not indicate form, but the pathologist uses this term to describe a type of carcinoma occurring in ulcerative colitis, and this type has a fairly specific roentgenologic picture.

The constricting lesion is characteristically long. In 1 case such a carcinoma was $8\frac{1}{2}$ inches long (Fig. 1). This is the most commonly encountered form of carcinoma in ulcerative colitis and represents more than half the total number of carcinomas in this series. The edges of the bowel involved by carcinoma are ragged and irregular. The lumen of the bowel is narrowed, and its ragged and irregular channel contains numerous small, polyp-like projections (Fig. 2). The ends of the constricted segments are cone-shaped, and they contain an occasional sharp edge of advancing carcinoma. The constriction may be concentric or eccentric, but it is always irregular. There is usually no sharp delineation between carcinoma and adjacent mucosa involved with ulcerative colitis.

The annular lesions resemble the napkin-ring carcinomas of the colon seen in patients without chronic ulcerative colitis. These lesions tend to be shorter, the ends are sharper, and the mucous membrane has the structureless mucosal relief pattern of carcinoma (Fig. 3).

Sessile carcinomas (Fig. 4) are also similar to those encountered in patients without ulcerative colitis. Annular and sessile carcinomas represented 6 of the 37 carcinomas seen in this group of 29 cases. (Some of the patients had multiple carcinomas.) Polypoid carcinomas were un-

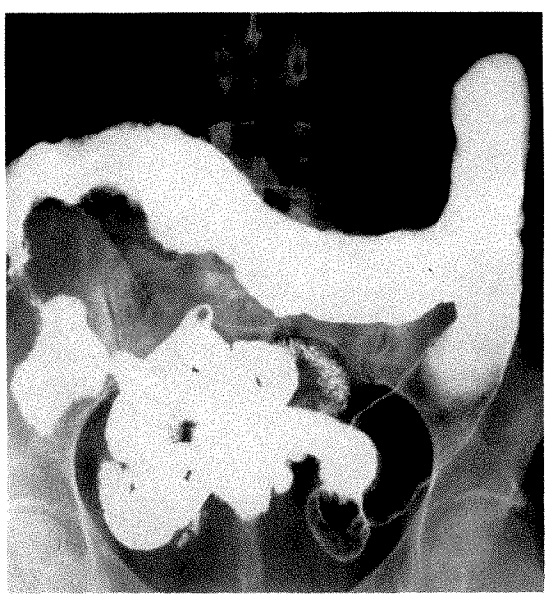


FIG. 1. Long carcinoma, involving the cecum, ascending colon, and transverse colon.

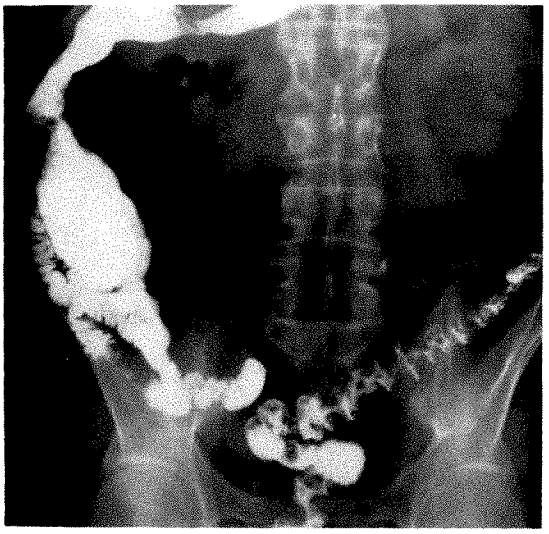


FIG. 2. Long carcinoma of the ascending colon, with multiple small polypoid defects in the lumen.

usual in this series; 4 pediculated polypoid carcinomas were observed. This form of carcinoma associated with ulcerative colitis may be more commonly seen by the proctologist.

So-called scirrhous carcinoma (Fig. 5) results in smooth, narrowed segments of colon without polypoid hyperplasia and without the ragged edges or irregular channel that is seen with the constricting lesions. In scirrhous carcinoma there may be no clinical or roentgenologic suspicion of neoplasm; these carcinomas may represent

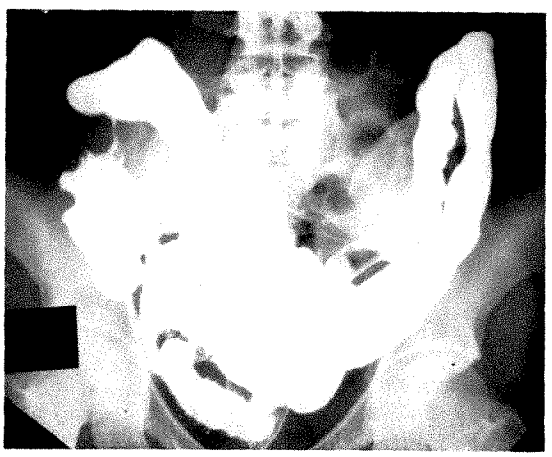


FIG. 3. Annular carcinoma in chronic ulcerative colitis.

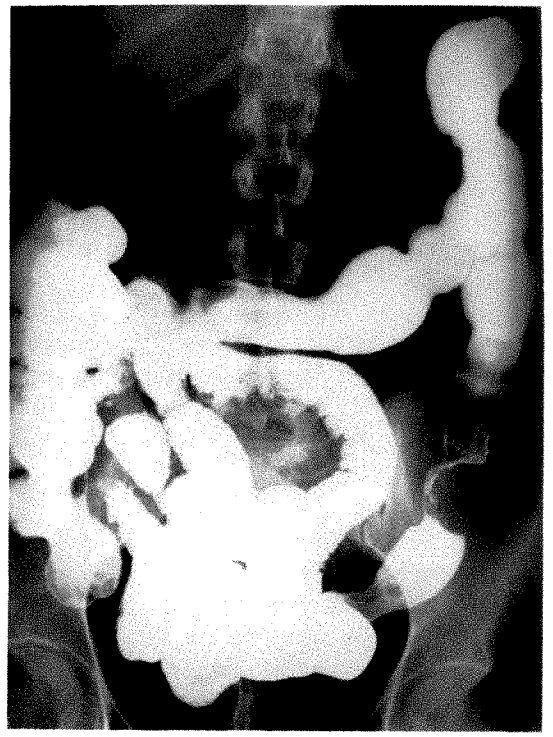


FIG. 4. Sessile carcinoma of the descending colon associated with chronic ulcerative colitis.

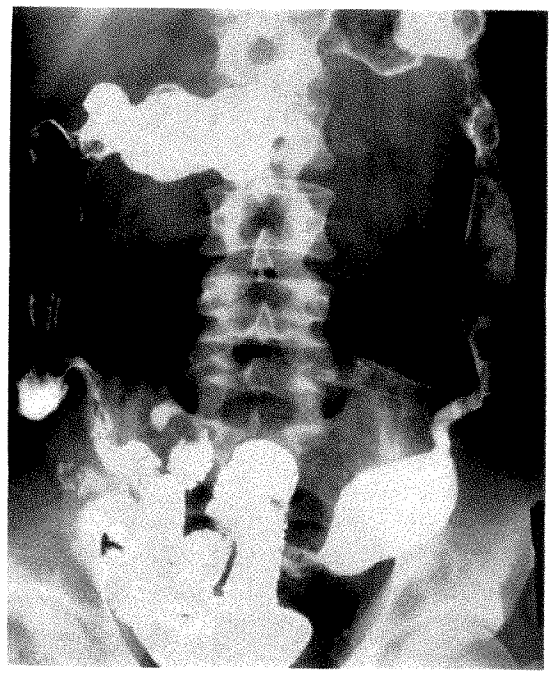


FIG. 5. Scirrhous carcinoma of the descending colon. Roentgenologically this is difficult to distinguish from stricture.

the "surprise cancer" found only at resection. Because of replacement of the mucosa by sheets of cancer cells, the bowel has a relatively flat surface which is indistinguishable roentgenologically from the denuded mucosa of burned-out chronic ulcerative colitis. When some segmental narrowing of the bowel is present, differentiation between carcinoma and stricture is difficult, since the flat surface of the lumen of the narrowed segment of bowel may look exactly the same in both scirrhous carcinoma and stricture.

In about half of our cases of carcinoma of the colon in chronic ulcerative colitis, the colitis was quiescent at the time of diagnosis of the carcinoma, and in most of the remaining cases only slight roentgenologic evidence of activity was present. However, carcinoma is not necessarily limited to those patients with contracted, nondistensible, hoselike colons (Fig. 6, *a* and *b*). Many patients with old, burned-out ulcerative colitis have some degree of distensibility of the colon. Distention of the bowel may reveal otherwise unsuspected lesions. For this reason fluoroscopy and roentgenograms of the barium-filled colon are important in the diagnosis of carcinoma.

Although obstruction did not occur in the present cases of carcinoma, it undoubtedly occurs occasionally in carcinoma associated with ulcerative colitis. The 2 cases of complete obstruction roentgenologically thought to involve carcinoma proved to represent strictures with no evidence of carcinoma. It may be that the scarring caused by ulcerative colitis prevents obstruction until the carcinoma is far advanced. The diagnosis then will usually have been made before obstruction occurs. In general, the lumen of the bowel at the region of carcinomatous growth is larger than that seen in carcinomas not associated with chronic ulcerative colitis.

In most cases the usual sharp delineation between carcinoma and normal bowel is not present. The margins of the carcinoma tend to fade off into the adjacent bowel already deformed by ulcerative colitis.

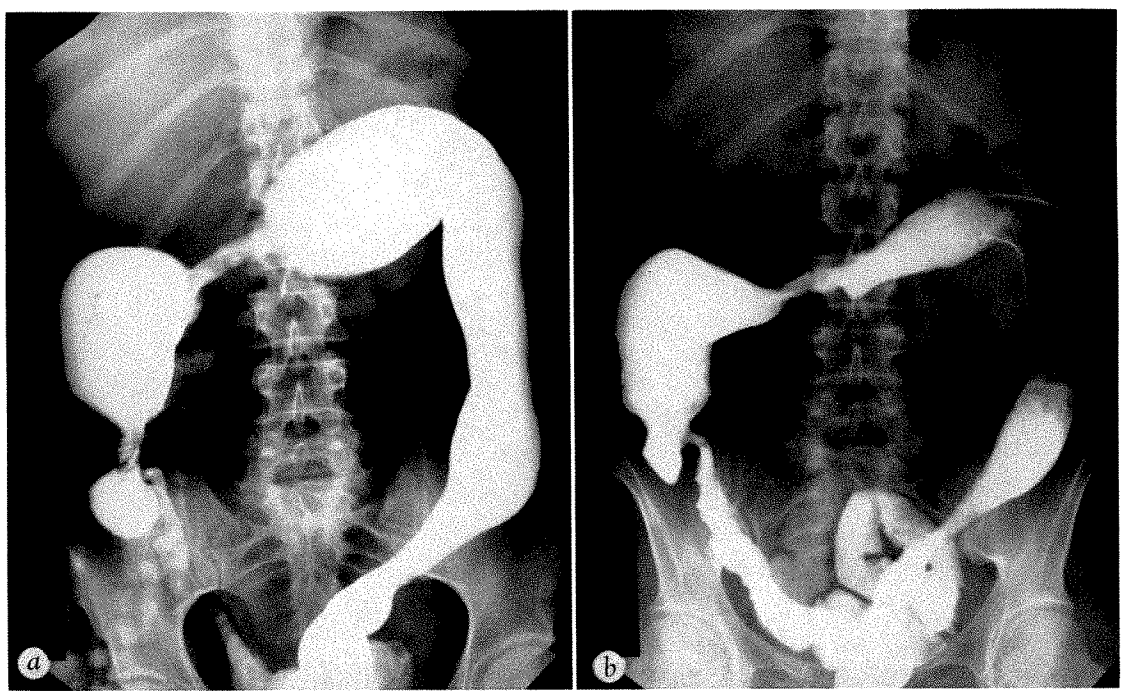


FIG. 6. (*a*) Carcinoma of the transverse colon. Note that distensibility of the colon is still present in this case of ulcerative colitis. (*b*) Postevacuation roentgenogram of *a*.

This is particularly true of the scirrhous variety of carcinoma.

Multiplicity of carcinomas is a fairly common finding in ulcerative colitis. Of the 29 patients in this series, 7 had multiple carcinomas. In most instances these multiple carcinomas were of the same morphologic type, although all different types may occur in the same patient. A higher incidence of multiplicity of carcinoma has been reported in ulcerative colitis,[4] but it must be considered that rectal lesions were excluded in the present study.

Roentgenologic evidence of activity of the ulcerative colitis is unusual. In the majority of instances the disease is quiescent. In about 25 per cent of cases carcinoma is associated with roentgenologic evidence of mild or moderate activity of the colitis. Conflicting opinion exists concerning the relationship of pseudopolypoid hyperplasia, polypoid hyperplasia, and the development of carcinoma in ulcerative colitis. The findings in the present series of 29 patients indicate that polypoid hyperplasia of the colon is less likely to be present in association with carcinoma if it is not localized in a segment of colon.

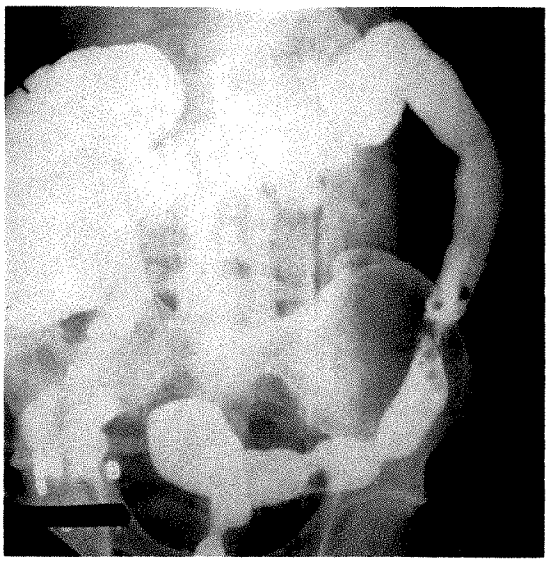


Fig. 7. Polypoid hyperplasia of the descending colon and sigmoid. No carcinoma is present.

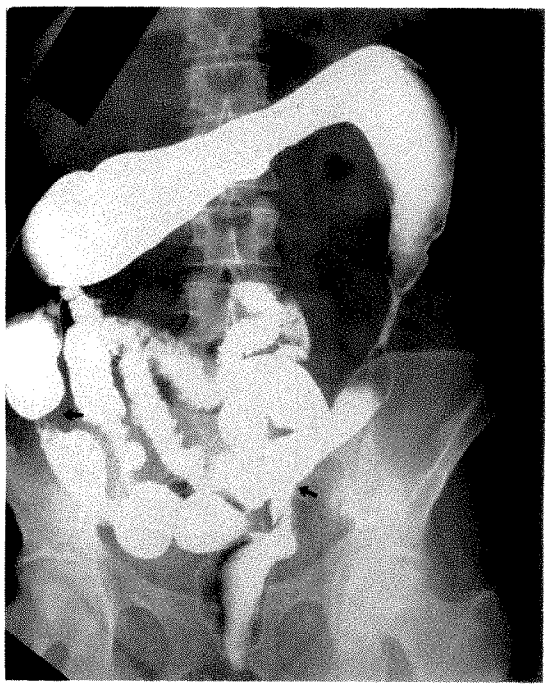


Fig. 8. Strictures (arrows) in the terminal ileum near the ileocecal valve and also in the descending colon. Carcinoma (scirrhous type) is present in the sigmoid.

DIFFERENTIAL DIAGNOSIS

The most common form of carcinoma associated with chronic ulcerative colitis—the long constricting carcinoma—must be distinguished from localized polypoid hyperplasia of the colon (Fig. 7). In general, the carcinoma will produce more ragged edges, a more irregular channel, and more constricting deformity. Because the changes differ only in degree, the differentiation between segmental polypoid hyperplasia and carcinoma may be difficult. However, none of the cases of simple polypoid hyperplasia showed the ragged, irregular destruction characteristic of carcinoma. With this type of lesion, diagnosis usually involves careful appraisal of the clinical status of the patient as well as roentgenologic evaluation. Often a definite diagnosis can be made only after surgical resection.

Annular, sessile, and polypoid lesions offer no serious problem in diagnosis, and they should be removed.

There seems to be no reliable way of determining the difference between benign stricture and so-called scirrhous carcinoma associated with chronic ulcerative colitis (Fig. 8). Close attention to the clinical status of the patient may be of some help. Exploration may be necessary for differentiation.

SUMMARY

The occurrence of carcinoma superimposed on ulcerative colitis is a well-established fact. Carcinoma tends to occur in younger persons with quiescent ulcerative colitis who have a history of the disease of about seventeen years' duration. In such cases the patient may complain of a recent change in bowel symptoms.

Roentgenologically, these lesions consist of constricting, sessile, polypoid, annular, and scirrhous defects. The most common form is the long constricting lesion with coned ends, ragged edges, small polyp-like defects in the lumen, and an adequate but ragged channel.

Differential diagnosis is difficult, particularly in the scirrhous variety, in which the lesions may often resemble strictures roentgenologically. Occasionally, resection of the colon will reveal a carcinoma which was not suspected clinically or roentgenologically.

Mayo Clinic
Rochester, Minnesota

REFERENCES

1. BARGEN, J. A. Chronic ulcerative colitis associated with malignant disease. *Arch. Surg.*, 1928, *17*, 561–576.
2. BARGEN, J. A., and DIXON, C. F. Chronic ulcerative colitis with associated carcinoma. *Arch. Surg.*, 1935, *30*, 854–864.
3. SAUER, W. G., and BARGEN, J. A. Chronic ulcerative colitis followed by carcinoma: report of twenty-six cases. *Proc. Staff Meet. Mayo Clin.*, 1944, *19*, 311–315.
4. SAUER, W. G., and BARGEN, J. A. Chronic ulcerative colitis and carcinoma. *J.A.M.A.*, 1949, *141*, 982–985.

CARCINOID OF THE RECTUM*

A CASE REPORT WITH OBSERVATIONS ON RADIOSENSITIVITY OF NODULAR METASTASES TO THE SKIN

By MERALL ROTH, M.D.†
SAN FRANCISCO, CALIFORNIA

CARCINOIDS are said to arise from argentaffin (Kultschitzky) cells normally present in the bases of the crypts of Lieberkühn in the gastrointestinal tract from the cardia of the stomach to the anus. Seventy to 90 per cent of the cases occur in the appendix. Most of the others are found in the ileum. The rectum is probably the most frequent site of origin in the colon. Multiple primary lesions are not uncommon, particularly in the small intestine. They have been found in patients ten days to eighty-nine years of age. Statistical data are summarized by Ritchie.[13] De Lashmutt and Kolozsi[3] found that the average age of patients with this tumor was forty-five years. Green[7] reports that 3 of his 6 cases ranged from twenty-one to thirty-two years in age.

Except for the carcinoids arising in the appendix where symptoms resembling the initial stages of appendicitis lead to early discovery, metastases are often quite large before a small primary lesion is found.[11] The metastatic spread occurs through the lymphatics of the mesentery to the regional lymph nodes and by the portal circulation to the liver. Metastases also have been reported in the bone, brain, skin and viscera. In 1944, in a survey of the literature, Ritchie and Stafford[14] found that only 12.6 per cent of 111 cases of rectal carcinoids had metastasized. This was the lowest incidence save for carcinoids of the appendix. These figures would have undoubtedly been revised upward with adequate follow-up. Survival for more than ten years is not uncommon and metastases from the rectum which were not grossly evident on an earlier observation may well appear later. Dockerty[4] states that nearly 50 per cent of rectal lesions larger than 5 mm. when first seen will metastasize. He also states that 5 per cent of submucosal nodules in the rectum seen by the proctoscopist are carcinoids. They are usually small, freely movable, and respond to fulguration. About 75 per cent of them are asymptomatic and are found incidentally. Symptomatic patients may have bleeding, diarrhea, constipation, and tenesmus.

Masson, forty years ago, referred to these tumors as argentaffinomas and insisted on the endocrine activity of the enterochromaffin cells. This was regarded as unfounded speculation. Since 1930, case reports appeared in the literature noting the coincidence of pulmonary stenosis and metastasizing carcinoid, but it was not until 1953 that two independent reports[9,15] established a new syndrome linking hormonal effects and cardiovascular abnormalities with the tumor. Since then other cases have been published.[10,18] In 1936, Erspamer[5] and his co-workers extracted a substance, which he called "enteramine" from tissues containing Kultschitzky or similar chromaffin cells. In 1952, this was identified as 5-hydroxytryptamine by Erspamer and Asero.[6] Page called this "serotonin."[12] It is now evident that some carcinoid tumors produce an excess amount of this substance causing violaceous cyanosis and telangiectasia of the face, episodic flushing, and at times tachycardia, hypotension, asthma, diarrhea, and abdominal pain. As it passes through the right heart, it apparently has a toxic effect on the endo-

* From The Department of Radiology, Veterans Administration Hospital, San Francisco, California.
† Chief, Radiology Service, Veterans Administration Hospital, San Francisco, California and Assistant Clinical Professor, Department of Radiology, University of California.

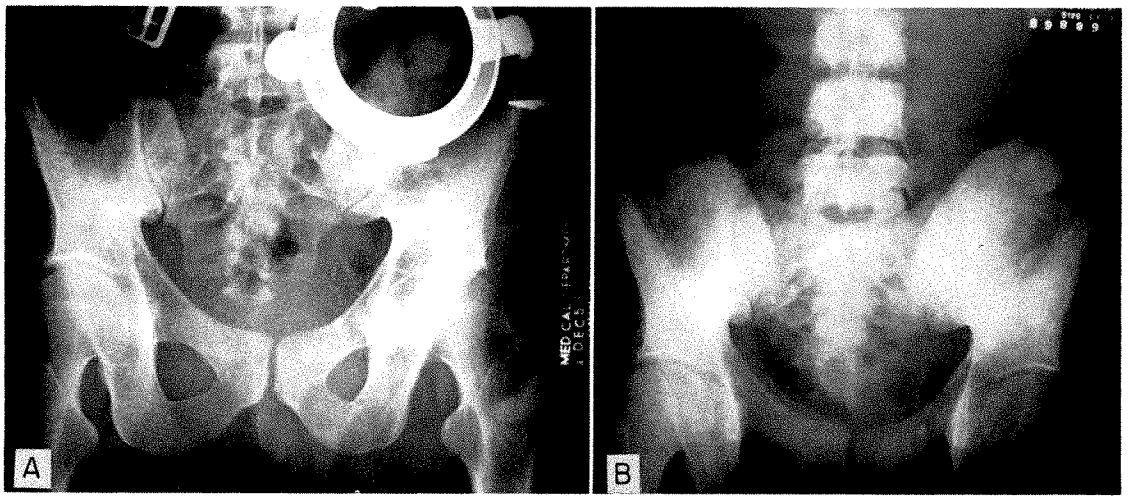


Fig. 1. Roentgenograms of the pelvis. (*A*) December, 1953. Early evidence of osteoblastic metastases was manifested by dense irregular trabeculations. These are seen in the pelvis. Note the colostomy. (*B*) April, 1956. The osteoblastic metastases progressed to dense homogeneous involvement of the lumbar spine and most of the pelvis. The process is less advanced in the pubic arches and upper femora. These changes were typical for the entire axial skeleton except for the skull which showed no metastatic lesions.

cardium, resulting in right heart failure from a fibrous endocarditis and valvulitis of either or both the pulmonic and tricuspid valves. For the most part, serotonin is carried by the platelets and is apparently inactivated by the lungs, sparing the left heart. It is excreted in the urine as 5-hydroxyindole-acetic acid.

REPORT OF A CASE

G.B. In October, 1948, this thirty-eight year old white male truck driver had three episodes of bloody bowel movements with a total loss of about a pint of dark red blood. Three weeks later he again had three bowel movements mixed with blood. A private physician found a small rectal lesion on a sigmoidoscopy. This was biopsied and reported as carcinoma.

The patient was referred to Oak Knoll Naval Hospital in Oakland in December. A 1 cm. papilloma on the anterior rectal wall 10 cm. from the anus was visualized through the sigmoidoscope. A biopsy was reported as carcinoid. An abdominoperineal resection was done on December 28, 1948. The rectum and sigmoid were removed. No gross lymphadenopathy was observed and the liver was normal to palpation. An ulcerated lesion, 17 mm. in diameter, with an elevated border was found in the rectum. Sections did not indicate any invasion of the muscularis. The pathologist was uncertain of the histology but thought it was a melanosarcoma. He noted that no chromaffin cells were found on special stains. It was reported as a malignancy, type undetermined. The patient was discharged feeling well with a functioning left lower quadrant colostomy. He returned to his former occupation and had no complaints until August, 1953 when he began to lose weight, became easily fatigued, and noted anorexia, as well as back and leg pain. Roentgenograms in December, 1953 showed dense irregular trabeculations in the lumbar spine and pelvis (Fig. 1*A*).

He was first seen in this hospital in March, 1954 and, at that time, was thoroughly investigated. It was felt that the patient had widespread metastatic bone disease (Fig. 2*A*). A biopsy of a skin nodule on the face revealed metastatic carcinoid. Bone marrow biopsies at that time were negative. Severe low back pain began in October, 1954. At that time the alkaline phosphatase was elevated to 16.1 King-Armstrong units. The hemoglobin was 12 gm. and the white blood cell count was 9,500 with a normal differential. Thioguanine was started in November and continued for eighteen months in the hope of arresting the growth of neoplasm. Initially, the dose was 120 mg. per day but, because of bone marrow depression requiring several transfusions, it was gradually

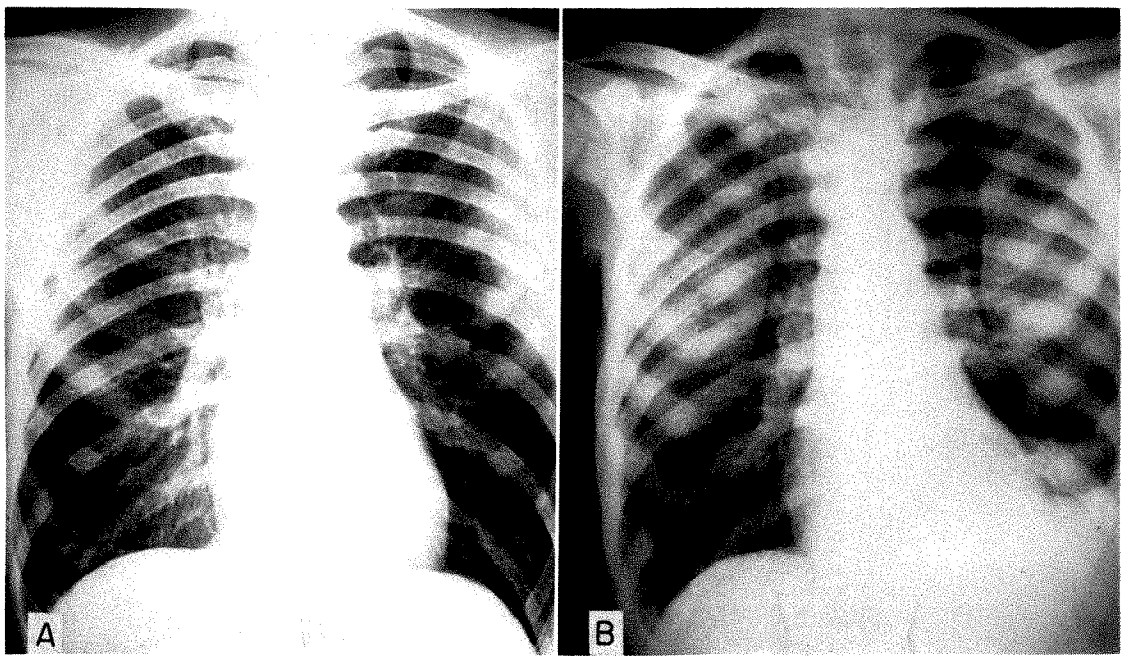


Fig. 2. Roentgenograms of the chest. (*A*) April, 1954. The osteoblastic process had diffusely infiltrated the ribs and thickened the trabeculae. Similar changes were found in the spine and pelvis. (*B*) April, 1956. The density of the ribs had increased markedly, and many of them are expanded. Fluid had collected at the left base.

reduced to 75 mg. per day. There was a gradual downhill course noted through several interval admissions. The diffuse osteoblastic metastases progressed unabated. Bone pain required continuous use of narcotics. Increasing numbers of skin nodules, up to 15 mm. in diameter, appeared on the face, scalp, arms and trunk. Some of these caused discomfort and were excised, confirming the diagnosis of metastatic carcinoid.

The final admission was in April, 1956 for edema, ataxia, numbness of the lower extremities and unremitting severe low back pain. The liver was four fingerbreadths below the costal margin. Thioguanine was discontinued since it was obviously ineffective. During this hospital stay there was increasing paresis, rising to the midabdomen. Bilateral Babinski signs appeared, and there was weakness of the small muscles of the hand. Clinically, it was felt that the patient had multiple metastases to the lumbar and cervical cord, involvement of the cauda equina and the lumbosacral plexus, and venous obstruction. A myelogram was unsuccessful. Roentgenograms of the spine, pelvis, humeri, femora and chest showed continued progression of the osteoblastic metastases (Fig. 1*B* and 2*B*). No metastases were visible in the skull. Fluid had collected at the left lung base. Laboratory data were as follows: spinal fluid slightly xanthochromic with 101 red blood cells, 1 lymphocyte, and 496 mg. per cent total protein; urine negative for melanin and serotonin; total serum protein 5.3 gm. per cent, albumin 3.0 gm. per cent, globulin 2.3 gm. per cent. The complete blood cell count on June 28 was hemoglobin 6.1 gm., white blood cells 4,450, differential normal, platelets 37,000, hematocrit 22.5.

He was referred to the radiologic department for palliative irradiation of the lumbosacral region for relief of back pain, venous obstruction and nerve root compression. A review of the literature gave no information regarding the radiosensitivity of this neoplasm, other than that it responded poorly to irradiation. Radiation therapy was begun on May 17 and continued for twenty-four days. A tumor dose of 2,100 r (half value layer 3 mm. Cu) was administered at a depth of 6 cm. The air dose was 2,380 r, and the skin dose was 3,150 r. No significant relief of pain was achieved and there

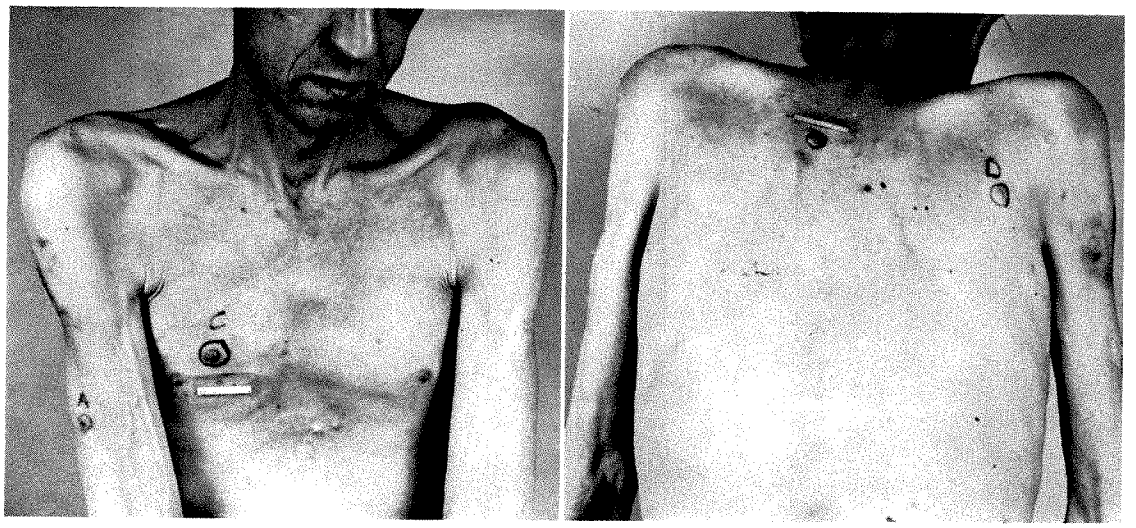


FIG. 3. May, 1956. Multiple skin nodules up to 15 mm. in diameter.

was no effect on the venous obstruction or on the neurologic changes.

A time dose study was undertaken on some of the nodules (Fig. 3) to determine the disappearance dose of irradiation, but this had to be abandoned shortly after it was under way because the deteriorating condition of the patient did not permit its continuation. The data collected are shown in Table 1 and Figure 4.

The first convulsion occurred on June 11. This was accompanied by apraxia and apnea, indicating cerebral metastases. Dilantin was started but this did not prevent recurrence. The patient continued to decline and expired on July 2, 1956, after an eight year history. No autopsy was obtained.

DISCUSSION

Dockerty's[4] comment regarding the high incidence of metastases from rectal lesions larger than 5 mm. was applicable in this case. About three weeks before the abdominoperineal resection, the lesion was a 1 cm. superficial papilloma. Although no metastases were found at surgery or in the specimen, five years later there was clinical evidence of this, confirmed by biopsy of skin nodules and roentgenographic evidence of diffuse metastases to bone. The dense homogeneous character of the osteoblastic process without a lytic component progressing from thickened, somewhat irregular, trabeculae is indeed interesting and attests to a slow stimulation of bone growth by the tumor. No serotonin was observed.

There is no definitive information in the literature regarding the radiosensitivity of this tumor. The multiple skin nodules provided an excellent opportunity for time dose studies but, unfortunately, the terminal status of the patient did not allow us to obtain any useful data for more than six days. Most of the observations in Table 1 and Figure 4 are for a one day period. The

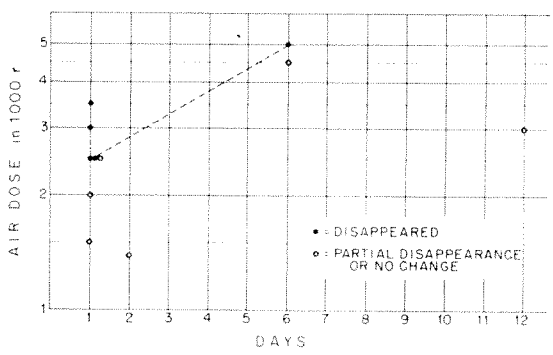


FIG. 4. Observation on the radiosensitivity of the skin metastases. The minimum disappearance dose for one treatment was 2,500 r in air. The data are inadequate for fractional doses. The minimum disappearance dose for six days may be 5,000 r in air.

TABLE I

OBSERVATIONS ON RADIOSENSITIVITY OF SKIN METASTASES

Nodule	Location	Lesion Diameter (mm.)	Field Diameter (mm.)	Half Value Layer (mm. Cu)	Fractions	Air Dose per Fraction	Total Treatment Time (days)	Total Air Dose	Last Observation after Final Treatment (days)	Skin Erythema	Comment
A	right arm	7.5	10	1.72	2	500	3	1,000	41	None	no change
B	posterior thorax	10	15	1.72	3	1,000	12	3,000	35	moderate 1°	no change
C	right pectoral	11	15	1.72	1	1,500	1	1,500	47	None	no change
D	posterior thorax	12	15	1.72	1	2,000	1	2,000	24	Moderate 2°	no change
E	posterior thorax	15	15	1.72	2	710	2	1,420	41	moderate 1°	no change
F	left shoulder	15	15	1.72	2	570	7	1,040	35	mild 1°	no change
G	posterior cervical region	8	10	1.72	1	2,500	1	2,500	33	Mild 1°	partial regression
H	cervical region	10	20	.57	1	2,500	1	2,500	28	moderate 2°	disappeared
I	posterior thorax	7	20	.57	1	3,000	1	3,000	28	mild 1°	disappeared
J	abdomen	7	20	.57	4	1,250	6	5,000	23	moderately severe 1°	disappeared
K	abdomen	10	20	.57	4	1,125	6	4,500	23	moderately severe 1°	small residual
L	sternum	5	20	.57	1	2,500	1	2,500	27	moderate 2°	disappeared
M	left arm	5	20	.57	1	3,000	1	3,000	25	moderate 1°	disappeared
N	left lumbar region	10	20	.57	1	3,500	1	3,500	23	moderately severe 1°	disappeared

minimum disappearance dose of 2,500 r for one day and 5,000 r for six days indicates that carcinoid is at least as radioresistant as squamous cell carcinoma and probably as resistant as adenocarcinoma. Cure, therefore, of visceral tumors by irradiation cannot be expected, even at the hazard of doing irreparable damage to normal tissues.

SUMMARY

A case of carcinoid of the rectum is presented. The patient was asymptomatic for five years following an abdominoperineal resection after which there were widespread metastases to bone, skin, viscera, spinal cord, and brain, respectively, with a fatal termination after eight years. Interesting features were dense osteoblastic metastases to the axial skeleton (skull excepted) and multiple skin nodules. Radiosensitivity studies on some of the nodules demonstrated a minimum disappearance dose of 2,500 r in air for one day and 5,000 r in air for six days.

The literature for carcinoids is reviewed briefly with particular reference to rectal lesions and to interesting hormonal effects exhibited by some of these tumors.

14 Via Cheparro Drive
Greenbrae
San Rafael, California

REFERENCES

1. BIÖRCK, G., AXEN, O., and THORSON, A. Unusual cyanosis in boy with congenital pulmonary stenosis and tricuspid insufficiency. Fatal outcome after angiocardiography. *Am. Heart J.*, 1952, *44*, 143–148.
2. BROWN, C. H., BISSONNETTE, R. P., and STEELE, H. H. Argentaffine tumors of gastrointestinal tract; report of eleven cases. *Gastroenterology*, 1949, *12*, 225–233.
3. DELASHMUTT, R. E., and KOLOZSI, W. A. Car-

cinoid tumors (argentaffinomas) of gastrointestinal tract: report of twelve cases. *Gastroenterology*, 1954, *26*, 743–753.

4. Dockerty, M. B. Carcinoids of gastrointestinal tract. *Am. J. Clin. Path.*, 1955, *25*, 794–795.
5. Erspamer, V. Die enterochromaffinen Zellen der Gallenwege in normalen und pathologischen Zuständen. (Nach Untersuchungen beim Menschen und bei Säugetieren.) *Virchows Arch. f. path. Anat.*, 1936, *297*, 70–92.
6. Erspamer, V., and Asero, B. Identification of enteramine, specific hormone of enterochromaffin cell system, as 5-hydroxytryptamine. *Nature*, London, 1952, *169*, 800–801.
7. Green, R. E. Carcinoid tumors of rectum. *Am. J. Surg.*, 1956, *91*, 351–357.
8. Horn, R. C., Jr. Carcinoid tumors of colon and rectum. *Cancer*, 1949, *2*, 819–837.
9. Isler, P., and Hedinger, C. Metastasierendes Dünndarmcarcinoid mit schweren, vorwiegend das rechte Herz betreffenden Klappenfehlern und Pulmonalstenose—ein eigenartiger Symptomenkomplex? *Schweiz. med. Wchnschr.*, 1953, *83*, 4–7.
10. McKusick, V. A. Carcinoid cardiovasulcar disease. *Bull. Johns Hopkins Hosp.*, 1956, *98*, 13–36.
11. O'Sullivan, W. D., and Bowe, J. J. Carcinoids of appendix and gastrointestinal tract. *A.M.A. Arch. Surg.*, 1954, *68*, 153–166.
12. Page, I. H. Serotonin (5-hydroxytryptamine). *Physiol. Rev.*, 1954, *34*, 563–588.
13. Ritchie, A. C. Carcinoid tumors. *Am. J. M. Sc.*, 1956, *232*, 311–328.
14. Ritchie, G., and Stafford, W. T. Argentaffin tumors of gastrointestinal tract. *Arch. Path.*, 1944, *38*, 123–127.
15. Rosenbaum, F. F., Santer, D. G., and Claudon, D. B. Essential telangiectasia, pulmonic and tricuspid stenosis, and neoplastic liver disease. Possible new clinical syndrome. *J. Lab. & Clin. Med.*, 1953, *42*, 941–942.
16. Spain, D. M. Association of gastrointestinal carcinoid tumor with cardiovascular abnormalities. *Am. J. Med.*, 1955, *19*, 366–369.
17. Tavenner, M. C., Bacon, H. E., and Peale, A. R. Carcinoid tumors of rectum; report of seven cases. *J. Internat. Coll. Surgeons*, 1951, *16*, 265–284.
18. Thorson, A., Biörck, G., Björkman, G., and Waldenström, J. Malignant carcinoid of small intestine with metastases to liver, valvular disease of right side of heart (pulmonary stenosis and tricuspid regurgitation without septal defects), peripheral vasomotor symptoms, bronchoconstriction, and unusual type of cyanosis; clinical and pathologic syndrome. *Am. Heart J.*, 1954, *47*, 795–817.
19. Waldenström, J. Carcinoidtumorer och vasomotorik; ett påpekande. *Svenska lak. tidn.*, 1953, *50*, 690–692.
20. Waldenström, J., and Ljungberg, E. Studies on functional circulatory influence from metastasizing carcinoid (argentaffine, enterochromaffine), tumours and their possible relation to enteramine production. I. Symptoms of carcinoidosis. *Acta med. Scand.*, 1955, *152*, 293–309. II. Chemistry of carcinoid tumours. *Acta med. Scand.*, 1955, *152*, 311–331.

VOL. 86, NO. 1

MESENTERIC ARTERIOGRAPHY*

By ALEXANDER R. MARGULIS, M.D., *and* PETER HEINBECKER, M.D.
ST. LOUIS, MISSOURI

THERE has been little basic progress in the roentgenography of the gastrointestinal tract since Dr. Cannon introduced radiopaque contrast material into the gut of a cat. Although refinements of his basic methods have been made, all deductions as to the existence of an abnormality in the gastrointestinal tract roentgenographically still are dependent upon the detection of the lesion's effect on the lumen of the gut. Although dependable for demonstrating the shape and contour of the inner surface of the bowel, existing intestinal roentgenography fails to provide means of assessing the architecture and limits of the whole lesion. Consequently, roentgenologic inferences as to the specific cellular characteristics of a given lesion of the intestine can be little more than an intelligent guess based largely upon frequencies. This applies especially to the problem of differentiating benign from malignant ulcers of the stomach and benign polyps from polypoid carcinomas in the stomach and colon. In an attempt to learn more about the roentgenographic anatomic details of intestinal lesions, both neoplastic and inflammatory, we have been employing operative and isolated organ mesenteric arteriography.

Bierman *et al.*[1] succeeded in opacifying the celiac axis, the superior and inferior mesenteric arteries by transaortic intra-arterial catheterization of viscera. The procedure was unpredictable and dangerous. Ödman[5,6] visualized the superior mesenteric artery by transfemoral arteriography. The results of this procedure are difficult to interpret because of superimposition of the opacified vessels of the intestinal mesentery.

Schobinger, Blackman and Lin[7] and Schobinger[8] demonstrated vascular patterns of benign and malignant neoplasms of the colon and of diverticulitis.

This study was undertaken to develop and to evaluate intestinal arteriography as a method for differentiating neoplastic from inflammatory lesions of the gastrointestinal tract and for distinguishing benign from malignant neoplasms.

METHOD

A limited trial of Schobinger and his coworkers'[7] method was disappointing because of the insufficient vascular detail obtained with it. In order to achieve the necessary detailed opacification of the intestinal vessels, we have used a film cassette wrapped in sterile plastic, placing it directly under the bowel. Then the roentgenograms were made before and during the terminal period of the injection of 8 to 10 ml. of contrast material into the proper artery through a plastic catheter or needle. The contrast material we first used was a solution of 37.5 per cent of diacetamido-triiodobenzoate (hypaque). However after Grayson *et al.*[2] showed that 40 per cent methylglucamine diacetylamino-triiodobenzoate (renografin) was less toxic to the bowel than hypaque, we used renografin exclusively. In order to facilitate subsequent arteriography of the removed piece of gastrointestinal tract, the plastic catheter was left in the proper artery after the *in vivo* examination was done.

CASE MATERIAL

Mesenteric arteriography was performed during an operation 35 times and upon 48

* From The Edward Mallinckrodt Institute of Radiology and the Department of Surgery, Washington University School of Medicine, St. Louis, Missouri.
Studies supported by a United States Public Health Grant, A4397.
Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

TABLE I

LESIONS STUDIED BY MESENTERIC ARTERIOGRAPHY

		Lesion	No.
Operative Arteriography	Colon	Carcinoma	15
		Adenomatous polyps	6
		Villous adenoma	1
		Lipoma	1
		Mucocele of appendix	1
		Chronic ulcerative colitis	3
		Diverticulitis	1
		Multiple small arteriovenous malformations	1
	Stomach	Carcinoma	3
		Peptic ulcer	3
		Total	35
Arteriography on Specimens	Colon	Carcinoma	28
		Adenomatous polyps	2
		Lipoma	1
		Villous adenoma	1
		Chronic ulcerative colitis	6
	Stomach	Carcinoma	6
		Leiomyosarcoma	1
		Peptic ulcer	3*
		Total	48

* One also had antral gastritis.

stomachs or colons after their removal from the body during celiotomy (Table 1).

RESULTS

The operative arteriograms of the intact bowel usually showed better arterial opacification than did the isolated organ arteriograms and therefore were easier to interpret. The vascular pattern outlined in an organ *in vivo* did not differ significantly from the pattern observed after the same organ was isolated.

CARCINOMA OF THE COLON

In all instances, operative and resected specimen arteriography of colonic carcinoma demonstrated destruction of the normal arcades of intestinal vessels. These were replaced by abnormal channels which were tortuous, angulated, had an irregular lumen, and coursed through the lesion chaotically in many directions (Fig. 1, *A–D;* and 2, *A* and *B*). The irregularity of lumen consisted of sudden irregular widenings and narrowings. In many roentgenograms the vessels of the tumor were finer and narrower than those of the adjacent intestine. The arteriograms of all colonic carcinomas contained irregular, undelimited areas of opacification. These were either small patchy areas of radiodensity resembling lakes surrounding vessels or large uniform areas of laking in which discrete vessels could not be seen. The small perivascular lakes were seen on all the arteriograms of colonic cancer. The larger areas of laking were found in 8 of 15 carcinomas injected *in vivo* and in 8 of the 18 tumors studied following their removal. These areas of laking were not seen when

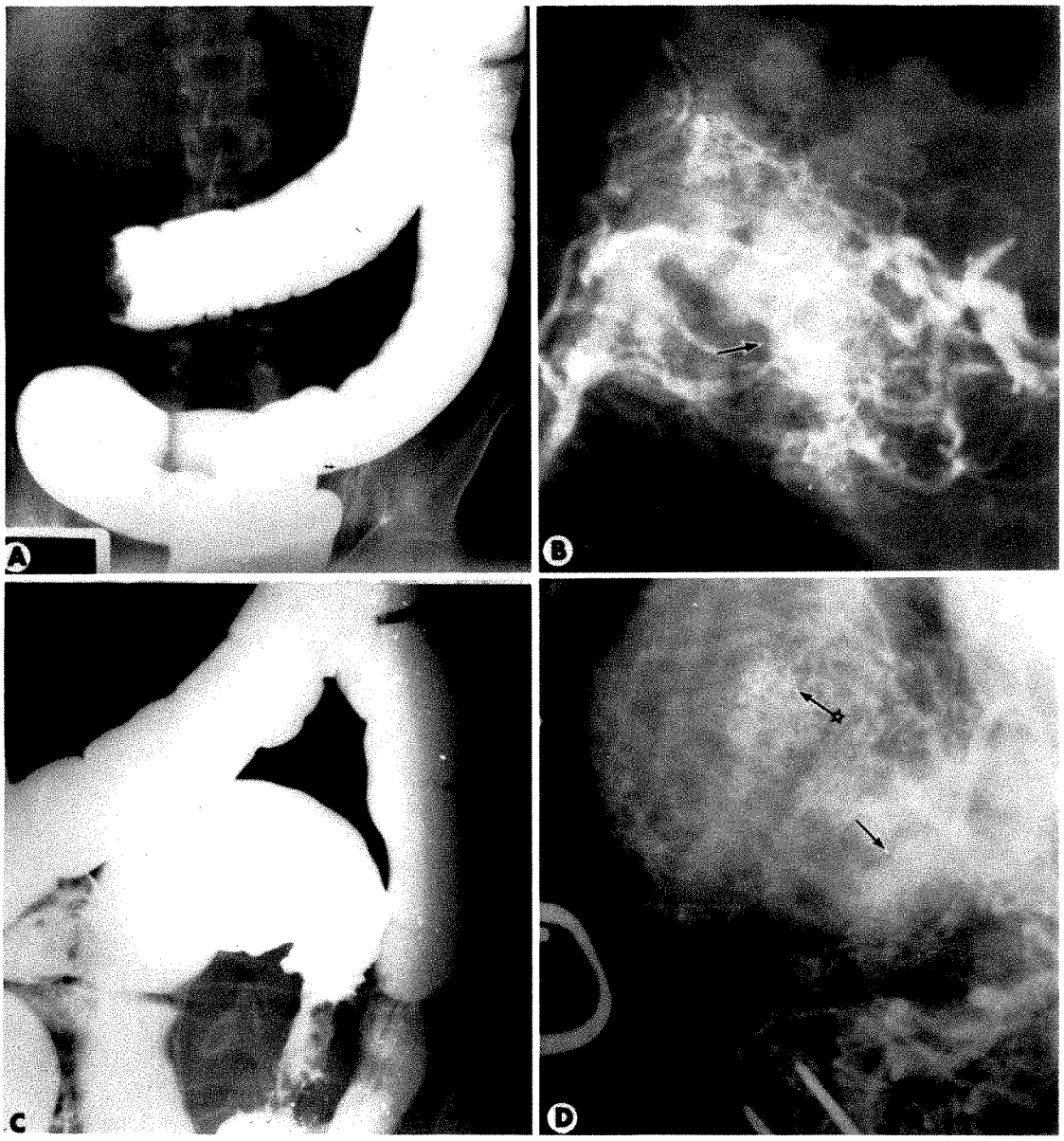


FIG. 1. (*A* and *B*) Annular carcinoma of the transverse colon. (*A*) A pre-evacuation barium enema roentgenogram showing the lesion. (*B*) An operative mesenteric arteriogram showing the lesion. The vessels are irregular and show sharp turnings. The blood vessels, in general, are more numerous than in the surrounding areas; however, close to the top of the lesion there are fewer vessels. Areas of dense opacification are seen in the tumor (arrow). (*C* and *D*) Carcinoma of the sigmoid colon. (*C*) Barium enema roentgenogram. (*D*) A mesenteric arteriogram, *in vivo*. Irregular, chaotically arranged vessels are seen throughout the tumor. There are areas of dense opacification (arrow) as well as smaller multiple perivascular areas of laking (arrow with star).

barium sulfate (USP) was used as the arteriographic contrast material in specimens.

The mesenteric arteries to the tumor always were prominent and numerous, often larger than adjacent mesenteric vessels. They originated from the marginal artery of the mesentery at irregular inter-

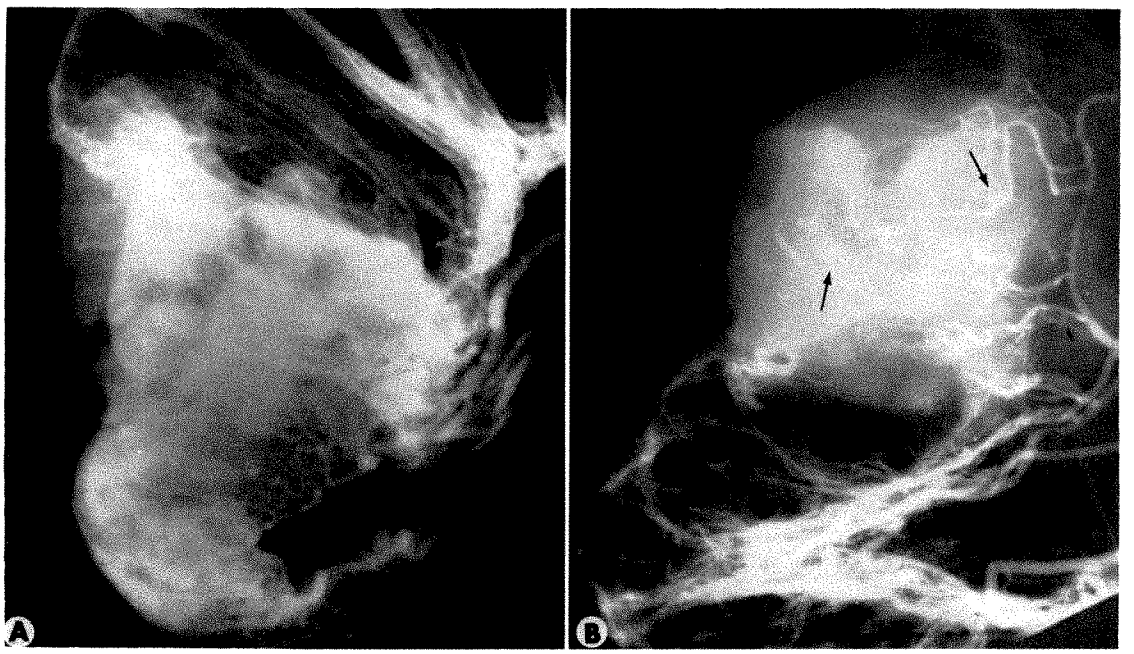


FIG. 2. (*A*) An operative mesenteric arteriogram showing a carcinoma of the cecum. Note that the mesenteric vessels to the tumor are quite large. The tumor shows large areas of laking along with multiple irregular vessels, sharply contrasting with the parallel normal vessels seen in the uninvolved bowel at the top of the figure. (*B*) Relatively avascular carcinoma of the sigmoid. The vessels in the tumor show an irregular lumen, sharp turnings and, in general, a chaotic direction. Arrows point to small areas of perivascular laking.

vals and often showed dilatations resembling aneurysms. The vascular channels visualized within the tumors were of smaller caliber and number than in the normal bowel wall in 3 of 15 cancers. Only 2 of the 15 carcinomas studied *in vivo* exhibited vessels uniformly more numerous than normal. The remaining 10 had areas in which only a few vessels were seen and other areas with numerous abnormal, opacified vessels.

BENIGN LESIONS OF THE COLON

Mesenteric arteriograms were performed in 6 patients being operated upon for adenomatous polyps. One had familial multiple polyposis. All others had one or more polypi that were larger than 2 cm. in diameter. One patient had a large villous adenoma (Fig. 3, *C* and *D*). The lesions in all 7 patients showed multi-directional vessels of larger caliber and number than normal. Looking with a magnifying glass at individual vessels within polyps on the arteriogram, luminal irregularities and sharp turnings were absent, although the vessels often were tortuous. The polyps did not contain diffuse or perivascular laking seen in cancers. All polyps except for the very small had a greater vascularity than did the normal adjacent intestine (Fig. 3, *A* and *B*).

Operative arteriograms of a mucocele of the appendix (Fig. 4, *A* and *B*) and of a colonic lipoma (Fig. 4, *C* and *D*) showed superficial vessels encircling the lesions but few if any within them. These arteriograms differed markedly from those of benign adenomatous polyps and of carcinoma.

The characteristics described serve as the basis for roentgenographic differentiation of benign from malignant colonic tumors (Fig. 1, *A–D*; 2, *A* and *B*; and 3, *A–D*) (Table II).

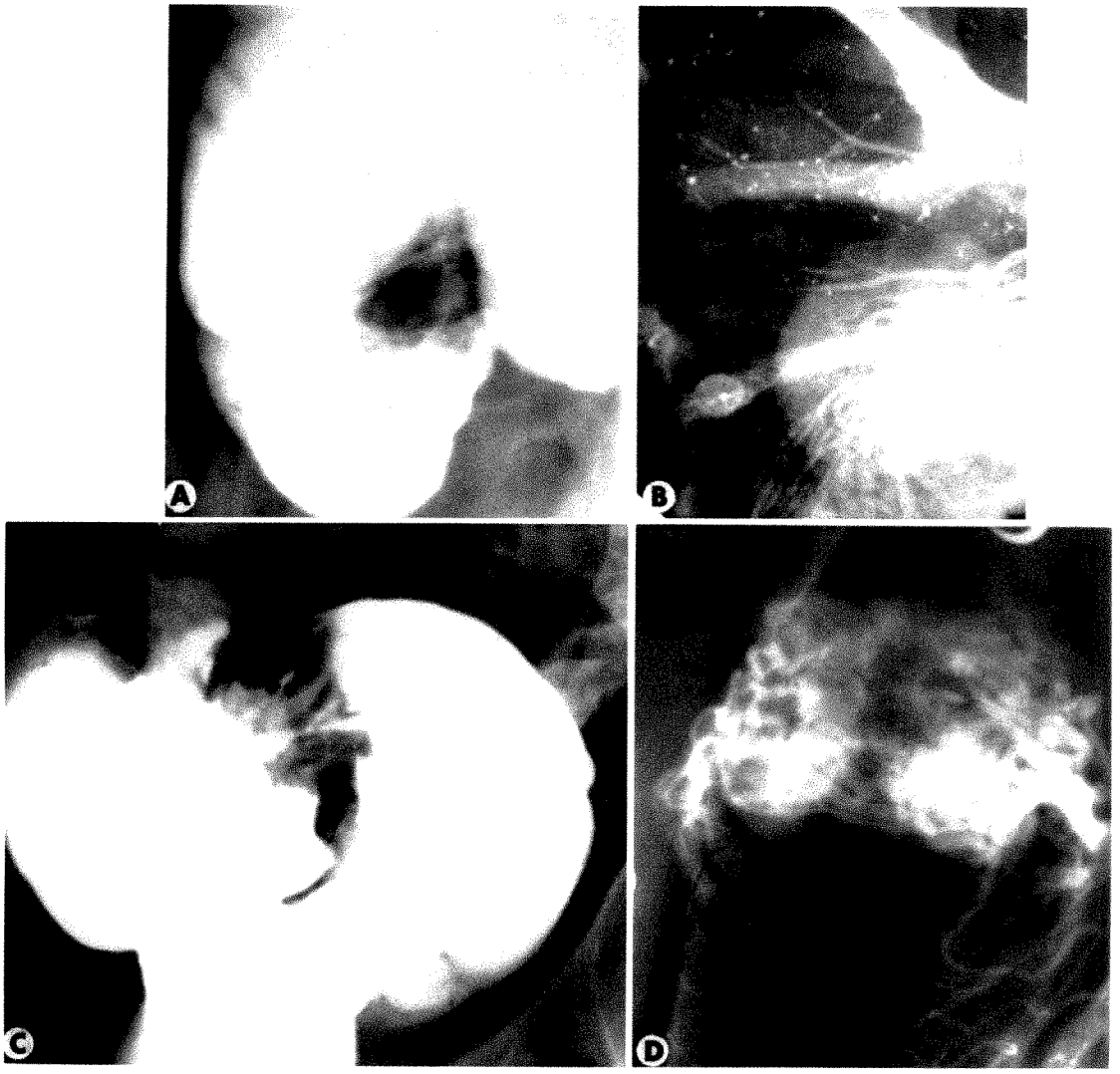


Fig. 3. (*A* and *B*) Adenomatous polyps of the cecum. (*A*) A spot roentgenogram during barium enema study showing the large filling defect in the cecum in the region of the ileocecal valve. (*B*) An operative mesenteric arteriogram showing one very large polyp and one smaller polyp. There are multiple winding vessels within the polyps but no areas of perivascular opacity or large areas of laking. The vessels fail to show irregular lumina and do not show sharp turnings. (*C* and *D*) Villous adenoma of the sigmoid. (*C*) A spot roentgenogram of the sigmoid obtained during a barium enema study. (*D*) An operative mesenteric arteriogram of the lesion showing numerous large tortuous vessels throughout the tumor, sharply contrasted with less numerous and smaller parallel vessels in the adjacent normal bowel. No areas of perivascular laking are seen.

Three patients with chronic ulcerative colitis had operative mesenteric arteriography. Six colons with the same disease were examined by arteriography after their removal. The colonic vessels were numerous, tortuous and gave off branches at close to a 90° angle. The normal parallel vessels forming arcades were replaced by the vessels characteristic of this disease. The difference in appearance, orientation and distribution from vessels in the normal bowel is illustrated in Figure 5*A*. Figure 5*B* is an operative arteriogram of another patient, containing the same type of vessels

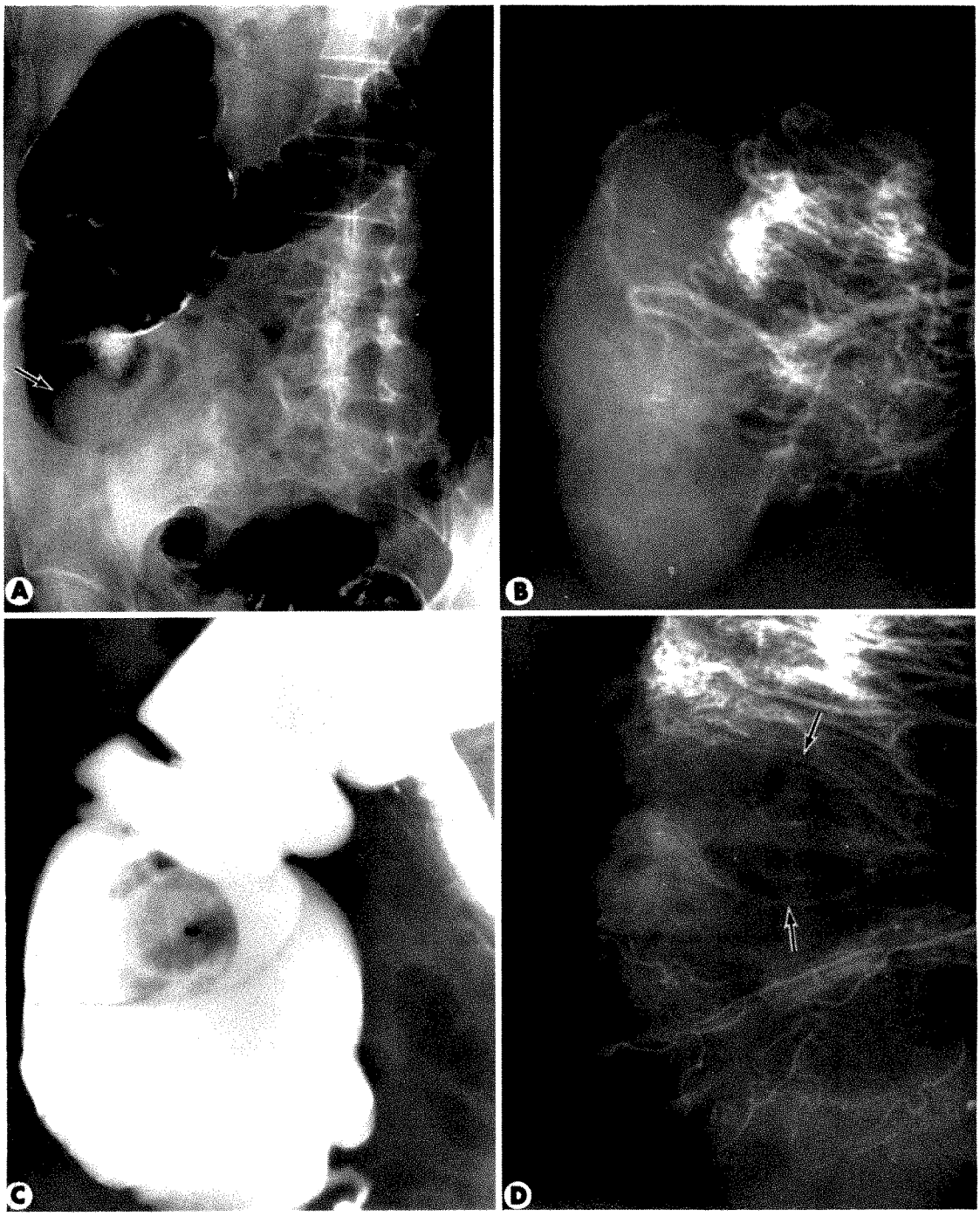


FIG. 4. (*A* and *B*) Mucocele of the appendix. (*A*) A large rounded filling defect is seen on the medial aspect of the cecum (arrow). (*B*) An operative mesenteric arteriogram shows the lesion to be relatively avascular. Only a few vessels are seen coursing on the surface of the tumor, in sharp contrast to the numerous vessels in the mesentery. (*C* and *D*) Lipoma of the cecum. (*C*) A spot roentgenogram of the cecum obtained during barium enema study shows a smooth rounded defect. (*D*) An operative mesenteric arteriogram shows the lesion to be radiolucent and relatively avascular. Only a few vessels are seen coursing on the surface.

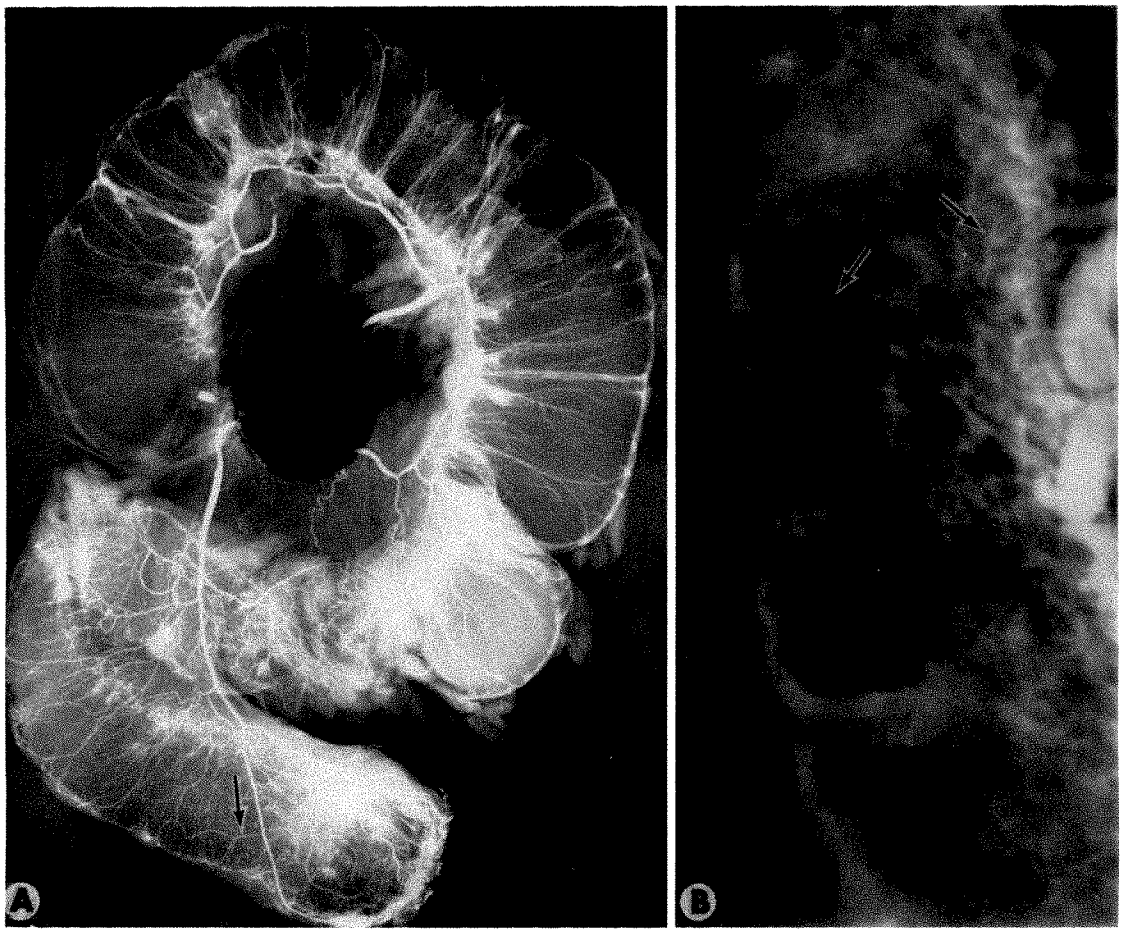


FIG. 5. (*A*) An arteriogram of the operative specimen following total colectomy. The distal colon is severely involved by chronic ulcerative colitis. The bowel wall is greatly thickened. The vessels are more numerous, tortuous and show branching at right angles (arrow). This is in sharp contrast to the normal arrangement of mesenteric vessels in the uninvolved bowel seen on the figure. (*B*) An operative mesenteric arteriogram in another case of chronic ulcerative colitis. Numerous tortuous vessels are seen. Many vessels show branching at right angles.

as seen in the specimen examination. The fecal stream had been diverted for a year in one case. This specimen showed only a few fine parallel blood vessels.

An operative arteriogram of a patient with diverticulitis of the transverse colon showed numerous dilated vessels in the area of inflammation just as seen by Schobinger.[8]

The vascular patterns of chronic ulcerative colitis and diverticulitis were different from that of malignant and benign neoplasms of the colon.

ARTERIOGRAPHY OF THE STOMACH

Operative arteriography of the stomach is not a formidable procedure. Anastomoses between arteries are abundant and injection of any vessels of the gastric arcades will visualize the area of abnormality. We have successfully studied lesions of the antrum, body and fundus of the stomach. Arteriograms of polypoid carcinoma of the stomach both *in vivo* and in the specimen showed the same type of vascular pattern described with the more vascular carcinomas of the colon. The normal paral-

TABLE II

FINDINGS IN MESENTERIC ARTERIOGRAPHY

	Lesion	Increased No. of Vessels in Diseased Area	Decreased No. of Vessels in Diseased Area	Areas of Increased and Decreased Vessels Side by Side	Large Areas of Laking	Perivascular Small Areas of Laking	Irregular Vascular Pattern	Vascular Luminal Irregularities	Changes in Directional Coursing of Vessels	Right Angle Branching
Colon	Carcinoma	±	±	Most common	In about 50%*	+*	+	+	+	o
	Adenomatous polyps	+	o	o	o	o	+	o	o	o
	Chronic ulcerative colitis	+	o	−	o	o	o	o	o	+
Stomach	Polypoid carcinoma	+	o	±	+	+	+	+	+	o
	Ulcerating carcinoma	o	+	o	o	o	o	+	o	o
	Peptic ulcer	+	o	±	o	o	o	o	o	o

* Except in specimen injected with USP barium sulfate.

lel arteries were absent. There were areas of diffuse and of small perivascular laking present (Fig. 6, *A–D*; and 7, *A* and *B*). The vessels in the tumor were irregular in lumen and showed sharp turnings in their course. Their direction was chaotic. Although certain areas of the tumor were relatively avascular, the vessels in general were more numerous. Arteries supplying the tumor were larger than those supplying normal areas.

Benign ulcers of the stomach showed numerous, wide, winding vessels surrounding the crater (Fig. 8, *C* and *D*). The immediate location of the crater and its margins may show few or no vessels. The vessels surrounding the ulcer crater were not irregular in either their lumen or distribution. Our series includes two malignant ulcers of the stomach. In both of them no evidence of cancer was detectable in the gross specimen and the cancer cells grew in sheaths surrounded by fibrous tissue. The diagnosis of carcinoma could be made only histologically. The arteriograms of the stomach following removal of the organ showed only a few vessels in the region involved by cancer. No areas of diffuse or perivascular laking could be demonstrated. The vessels appeared finer and smaller than in the arteriograms of benign peptic ulcer. The lumen of some of the vessels showed irregularly spaced, asymmetric areas of narrowing and dilatation. The distribution of the vessels, however, followed the normal pattern (Fig. 8, *A* and *B*). The difference between benign peptic ulcers and ulcerating carcinomas will have to be evaluated by a larger series.

In one case having antral gastritis with a benign peptic ulcer, the arteriogram showed numerous vessels of normal ap-

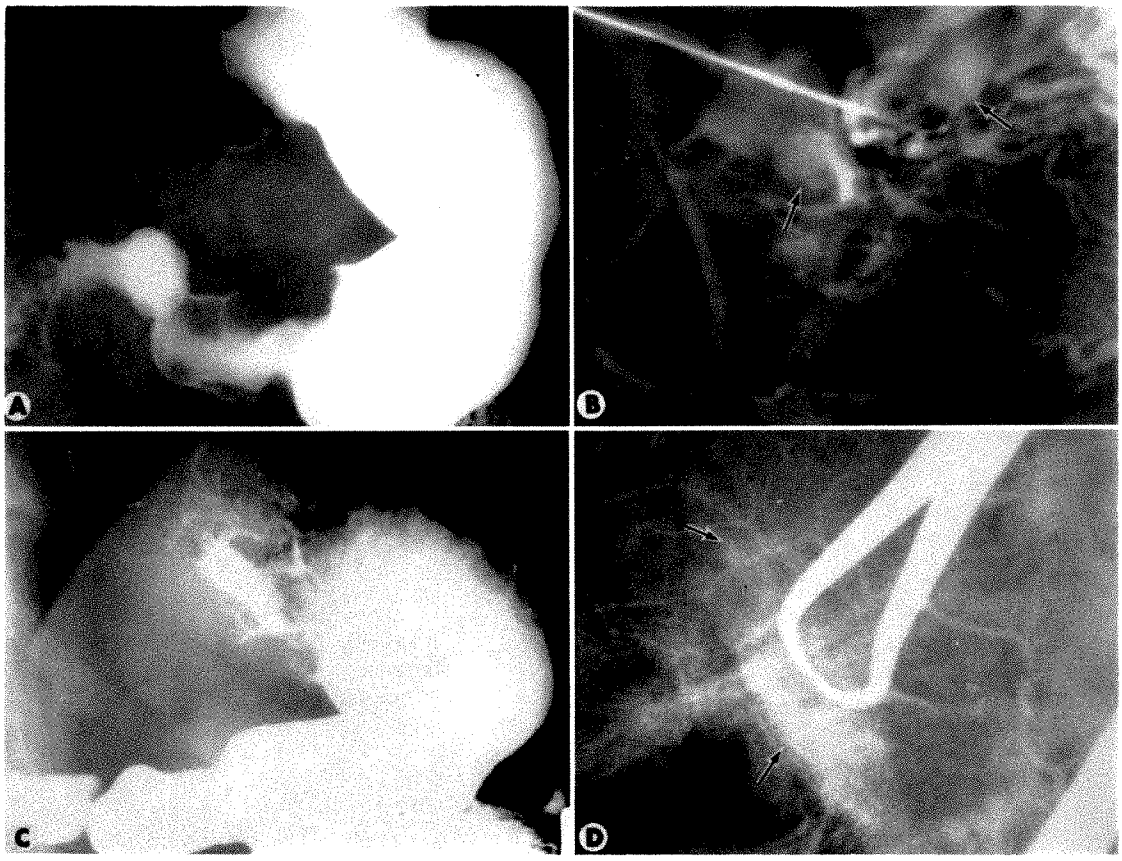


FIG. 6. (*A* and *B*) Polypoid carcinoma of the antrum of the stomach. (*A*) An upper gastrointestinal study with barium. (*B*) A mesenteric arteriogram with injection into the right gastric artery. Arrows point to areas of laking in the tumor. The vessels leading to the tumor are large and numerous. (*C* and *D*) Carcinoma of the fundus of the stomach. (*C*) An upper gastrointestinal study with barium. A large ulcer is seen within the mass in the fundus of the stomach. (*D*) An operative mesenteric arteriogram with injection into the left gastric artery shows areas of increased and decreased vascularity side by side. The vessels are irregular in distribution. Large areas of opacity in the tumor (arrow) are seen along with numerous small areas of perivascular laking (arrow with star).

pearance and distribution. An arteriogram of the specimen of a stomach containing a large leiomyosarcoma showed the same characteristics as seen in polypoid gastric carcinomas.

DISCUSSION

The definition of the location, extent and nature of internal lesions of the intestinal tract, without opening it, is surgically important. Operative arteriography promises to provide a fairly accurate way of doing this. The method, although seemingly simple and straightforward, is nonetheless somewhat time consuming. It often requires mobilization of the bowel or stomach in order to place the film cassettes in proper contact. It would be easier to have the film under the patient in a Bucky device but with the present technique this gives much poorer detail. Differences in the vascular pattern of a benign adenoma of the colon and a polypoid carcinoma are very fine, and any sacrifice in detail may lead to the wrong diagnosis.

It is difficult to draw final conclusions on the basis of studies done to date. Undoubtedly, much more work will be needed

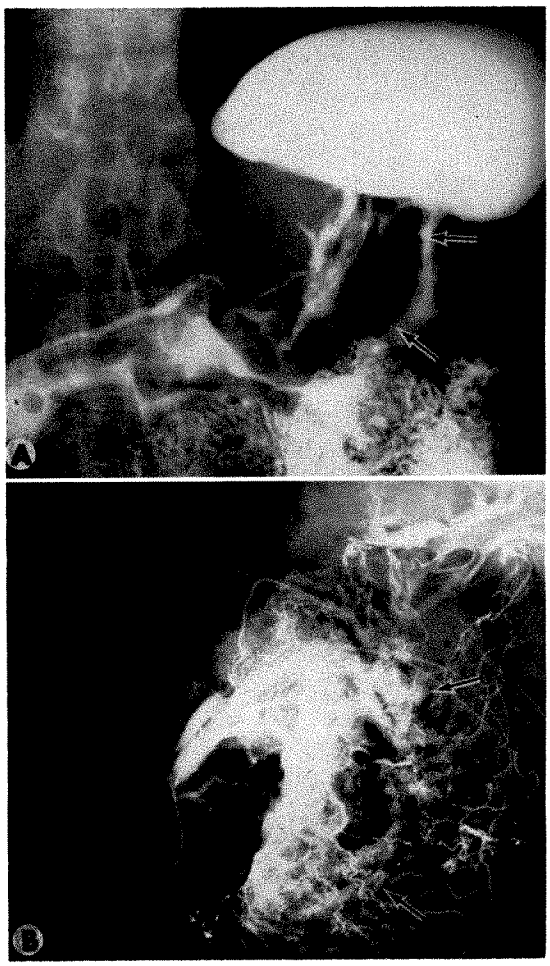


FIG. 7. Large polypoid carcinoma of the stomach. (*A*) A supine study with barium. The arrows point to the lateral margin of the mass. (*B*) An arteriogram of the dissected portion of the stomach. Within the tumor are numerous irregular vessels and large areas of opacification as well as those of perivascular density. The extent of the tumor is outlined by arrows.

before definite vascular patterns for various types of neoplasms of the stomach, small bowel and colon are established and before the validity of diagnostic criteria of this method survives repeated testing. The criteria arrived at from our material so far are given in Table II.

Besides the obvious utility of operative mesenteric arteriography for differentiation of various tumors and inflammations of the intestinal tract, it is, at times, very useful for the location of the site of massive gastrointestinal hemorrhage. We have already reported its use in this regard.[3] So far, we have used operative mesenteric arteriography only on the parts of the gastrointestinal tract that the surgeon has already decided to remove, having been worried about the toxic propensities of the contrast materials available. Evaluating the safety of this procedure in the dog, we[2] have found that methylglucamine diacetylamino-triiodobenzoate (renografin) is the safest contrast medium of those studied. Eight to ten milliliters of 40 per cent renografin in saline appear to be a safe amount to inject into a loop of bowel which is not to be resected.

SUMMARY

The method and findings of operative mesenteric arteriography are presented. Data obtained from injections of operative specimens of the colon and stomach are also referred to. Vascular patterns obtained by arteriography of malignant and benign tumors of the colon, chronic ulcerative colitis, carcinoma of the stomach and gastric ulcers are described.

Alexander R. Margulis, M.D.
Washington University School of Medicine
St. Louis, Missouri

REFERENCES

1. BIERMAN, H. R., MILLER, E. R., BYRON, R. L., JR., DOD, K. S., KELLY, K. H., and BLACK, D. H. Intra-arterial catheterization of viscera in man. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1951, *66*, 555–568.
2. GRAYSON, T., MARGULIS, A. R., HEINBECKER, P., and SALTZSTEIN, S. L. Safety of mesenteric arteriography; experimental study on dogs. To be published.
3. MARGULIS, A. R., HEINBECKER, P., and BERNARD, H. R. Operative mesenteric arteriography in search for site of bleeding in unexplained gastrointestinal hemorrhage; preliminary report. *Surgery*, 1960, *48*, 534–539.
4. MCAFEE, J. G. Survey of complications of abdominal aortography. *Radiology*, 1957, *68*, 825–838.
5. ÖDMAN, P. Percutaneous selective angiography

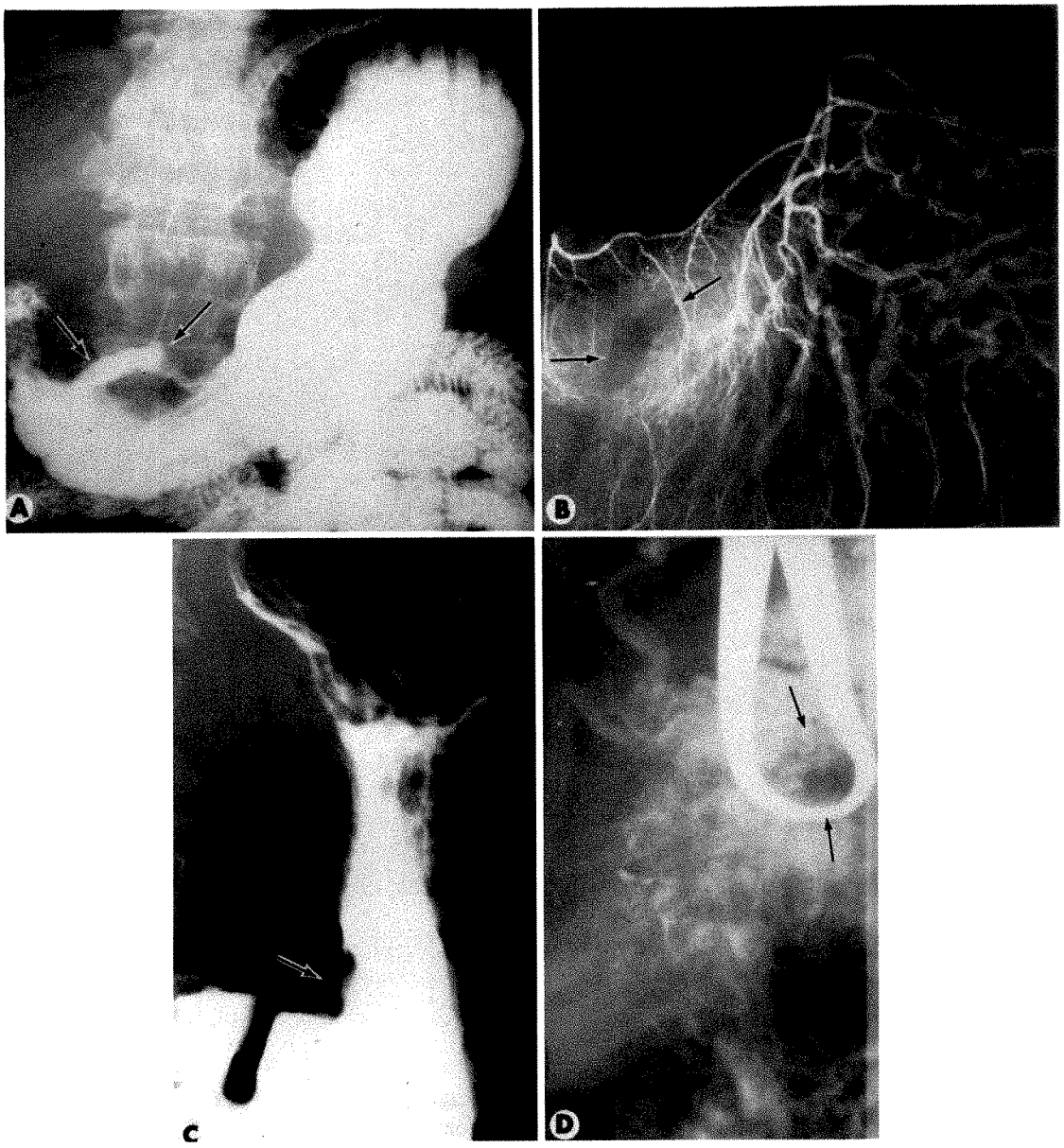


Fig. 8. (*A* and *B*) Large ulcerative carcinoma of the stomach. (*A*) A roentgenogram of the upper gastrointestinal tract with barium. (*B*) Only few vessels are seen in the area involved by the tumor. There is a strong suggestion of some irregularity in the lumen of the many attenuated vessels. (*C* and *D*) Benign peptic ulcer of the lesser curvature of the stomach. (*C*) An upright roentgenogram of the barium filled stomach with the ulcer shown in profile (arrow). (*D*) An operative arteriogram. Numerous tortuous vessels surround the ulcer which is the radiolucent area (arrows). There is no evidence of perivascular opacity. (A metallic instrument was left in the field of exposure by error.)

of coeliac artery. *Acta radiol.*, 1958, Suppl. 159.
6. Ödman, P. Percutaneous selective angiography of superior mesenteric artery. *Acta radiol.*, 1959, *51*, 25–32.
7. Schobinger, R., Blackman, G., and Lin, R. K. Operative intestinal arteriography. *Acta radiol.*, 1957, *48*, 330–336.
8. Schobinger, R. Operative intestinal arteriography in diagnosis of diverticulitis of colon. *Acta radiol.*, 1959, *52*, 28–33.

URETERAL OBSTRUCTION FROM REGIONAL ENTERITIS*

By C. JULES ROMINGER, M.D.,† RICHARD H. FLANDREAU, M.D.,‡ FRANCIS T. McGINNIS, M.D.,‡ *and* CHARLES SCHNALL, M.D.§
PHILADELPHIA, PENNSYLVANIA

URETERAL obstruction from regional enteritis is unusual. However, familiarity with this possibility can result in a correct preoperative diagnosis of enteritis, even in the absence of dominant intestinal symptomatology. The purpose of this paper is to report 2 cases illustrating this fact.

Urologic complications as a result of intestinal disease are well known.[8] They are usually caused by colonic disease, with abscess and fistula formation as the most common complications. Less well known is the fact that ureteral obstruction can occur as a result of regional ileitis without abscess formation.[7]

Hyams *et al.*[6] reviewed the literature to 1943, including over 1,000 cases of ileitis, and describe what is apparently the first case of ureteral obstruction from this disease.

Ginzburg and Oppenheimer[4] report 13 urologic complications in 300 cases of regional enteritis, but all of these were fistulae with abscess formation. Narath[8] mentions many extrinsic inflammatory causes of ureteral obstruction but not enteritis. Most urologic and gastrointestinal roentgenologic texts[1,2,5] do not mention the association of regional enteritis and ureteral obstruction. Redish[9] in 1952 reported 6 cases of ureteral obstruction due to regional enteritis. Two of these patients, who were known to have regional enteritis, presented with right urinary tract complaints. The other 4 cases were found upon survey study of a group of 22 patients with known enteritis and no urinary symptoms.

REPORT OF CASES

CASE 1. W.O'B., a thirty-three year old white woman, was admitted to Misericordia Hospital, Philadelphia, on April 22, 1957 complaining of right lower quadrant pain. The patient had been well until one month prior to admission when she developed mild, intermittent, colicky pains in the right lower quadrant of the abdomen which were associated with frequent, non-bloody, loose bowel movements and occasional nausea and vomiting. The positive physical findings were limited to the patient's abdomen. Tenderness associated with rebound and percussion tenderness were present in the right lower quadrant. There were no organs or masses palpable. Peristalsis was normal. Slight tenderness was present in the right flank. All laboratory examinations were within normal limits.

The right lower quadrant pain and tenderness gradually subsided during the first three days of hospitalization. An intravenous urogram revealed marked dilatation of the upper two-thirds of the right ureter associated with calyceal dilatation of the right kidney (Fig. 1). Retrograde urography demonstrated similar findings in addition to a partial ureteral obstruction at the level of the first sacral vertebra. A barium enema examination showed a large, rounded, filling defect in the right transverse colon just distal to the hepatic flexure (Fig. 2).

After suitable preparation, the patient underwent exploratory laparotomy on May 4, 1957 at which time the distal 20 cm. of the ileum was found to be involved in one large inflammatory mass. The bowel was markedly erythematous, edematous and friable. This large mass lay in the right paravertebral gutter and was obviously compressing and distorting the right ureter. The mass was also adherent to the right transverse colon. The terminal 20 cm. of ileum, along with the ascending colon and the right half of the transverse colon, was resected and an end-to-end ileotransversecolostomy was accomplished. The patient withstood this operative procedure well and subsequently had an uneventful recovery. A postoperative intravenous urogram revealed a normal right upper urinary tract. The pathologic report was regional enteritis.

* From the Departments of Radiology,† Surgery‡ and Medicine,§ Misericordia Hospital, Philadelphia, Pennsylvania.

Case II. N.U., a twenty-nine year old white woman, was admitted to Misericordia Hospital, Philadelphia, on September 24, 1959 complaining of right upper abdominal pain associated with nausea and vomiting. Two months prior to admission the patient, in her usual state of good health, experienced the first attack of crampy epigastric and right upper quadrant pain which radiated to the right subcostal area. An episode of diarrhea without melena occurred at this time and lasted for one week. Several similar attacks occurred during the ensuing weeks prior to admission. On the day before admission, following the ingestion of fried hamburger the patient experienced another episode of epigastric and right upper quadrant abdominal pain and vomited most of the ingested food. Chills and fever were present for two days prior to admission.

In the past the patient had undergone an appendectomy and inguinal herniorrhaphy. An undetermined type of "rectal operation" also had been performed some years previously. Upper gastrointestinal roentgenograms and a cholecystogram performed one month prior to admission at another institution were reported as normal.

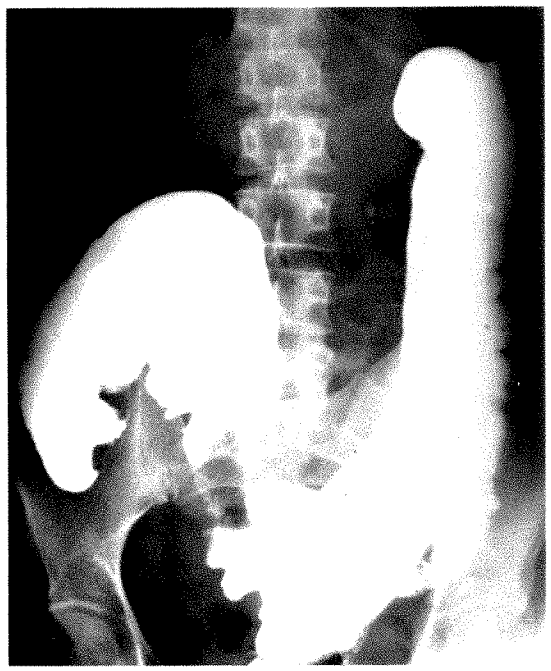


Fig. 2. Case 1. Barium enema examination demonstrates a filling defect in the proximal transverse colon in the right midsacral area, the same region as the ureteral obstruction.

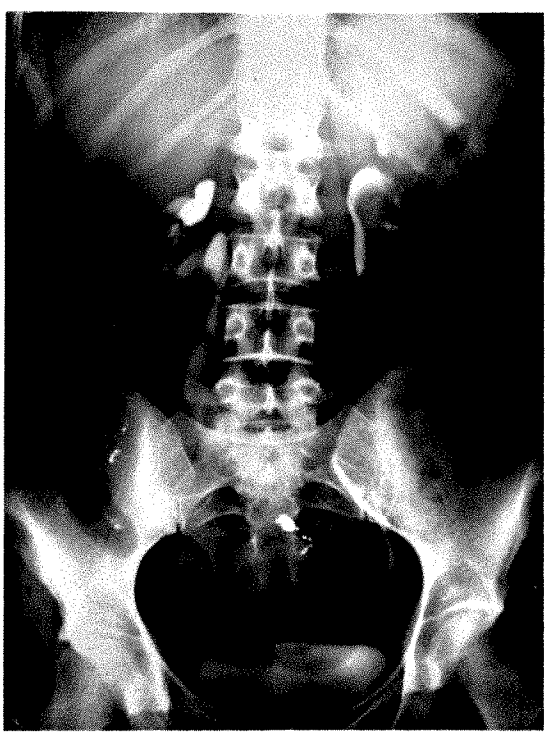


Fig. 1. Case 1. Intravenous urogram shows right ureteral obstruction at the level of the midsacrum.

The positive physical findings were limited to the abdomen, where the scars of previous surgery were noted. The abdomen appeared tense and somewhat rounded. Complete evaluation was difficult because of voluntary muscle guarding. There was moderate tenderness in the right upper quadrant and right periumbilical areas. There was slight tenderness in the right flank. No masses or organs were palpable.

During the first five days after admission the patient's fever ranged from 100° to 103° F. This gradually subsided spontaneously without specific treatment. Small doses of narcotics were necessary for the control of continued right upper quadrant and right periumbilical crampy pain. The tenderness persisted in these areas but was never acute.

Laboratory investigation revealed a normal hemogram and urinalysis. An upper gastrointestinal series and cholecystography showed findings within normal limits. Intravenous urography revealed a moderate distention of the calyceal system of the right kidney; the right ureter was dilated in the upper half and an area of constriction was visualized in the midureter (Fig. 3). This was thought to represent extrinsic compression. Right retrograde

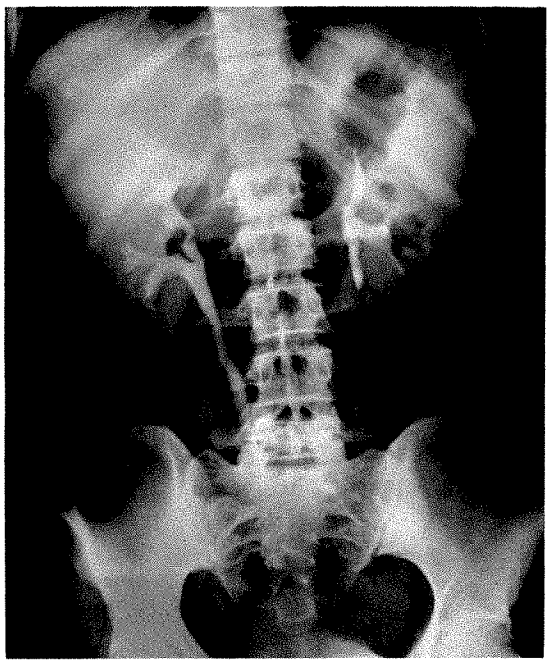


FIG. 3. Case II. Preoperative intravenous urogram shows a dilated right ureter down to the upper sacral area.

urograms confirmed the findings of an extrinsic pressure defect in the ureter at the level of the body of the fourth lumbar vertebra (Fig. 4 and 5). Considerable distention of the small bowel with numerous air-fluid levels was evident on plain roentgenograms of the abdomen (Fig. 6 and 7). A diagnosis of intestinal disease, possibly enteritis, causing urinary obstruction was suggested.

At laparotomy on October 12, 1959 approximately eighteen inches of adherent, inflammed and edematous bowel was found in the right paravertebral gutter in the region of the fourth lumbar vertebra. The adjacent mesentery was thickened, edematous and contained numerous enlarged lymph nodes. This large mass of inflammed small intestine and mesentery was intimately adherent to the right ureter, which resulted in its distortion and partial obstruction. Two additional "skip areas" of involvement, each measuring about six inches, were found in the proximal portion of the ileum. The small intestine proximal to these areas was edematous and dilated but not obviously inflammed. These dilated fluid-containing loops extended from the most proximal area of involvement up to the ligament of Treitz. The involved segment of ileum was excluded from the intestinal tract and a bypass ileotransversostomy was accomplished. The patient tolerated the operative procedure well and subsequently experienced an uneventful postoperative course.

Prior to the patient's discharge, intravenous urography revealed a normal right upper urinary tract (Fig. 8). The patient has enjoyed good health in the six month period since the operation. During this time she has been completely free of abdominal complaints and has gained five pounds in weight. An initial diarrhea of six to seven bowel movements daily has gradually subsided. Currently, the patient has one to two soft formed bowel movements daily. Intravenous urography revealed a normal urinary tract six months following operation.

DISCUSSION

Urinary tract involvement in intestinal disease is well known.[8] Most of the reported complications have been abscess and fistula formation and have been due to

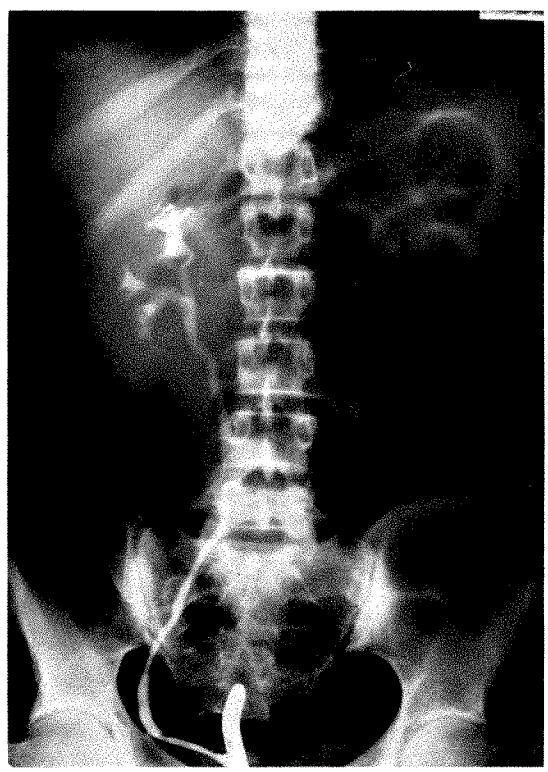


FIG. 4. Case II. Right retrograde urogram shows slight dilatation of the upper two-thirds of the right ureter. Note the distended small intestine in the right upper abdomen.

malignant disease and diverticulitis of the colon. Abscess and fistula formation as a result of regional enteritis are not common, occurring in about 3 per cent of large series of cases.

Ureteral obstruction as a result of regional enteritis without abscess formation is distinctly uncommon, but at least 7 cases have been reported.[4,6,9] In most of these cases the right ureter has been obstructed. In 1 case both ureters were involved. All of the previously reported cases were in men. Our 2 cases were in young women. In 3 of the reported cases, the patient presented with right urinary tract



FIG. 5. Case II. Right retrograde urogram in the oblique view showing constriction in the middle third of the ureter.

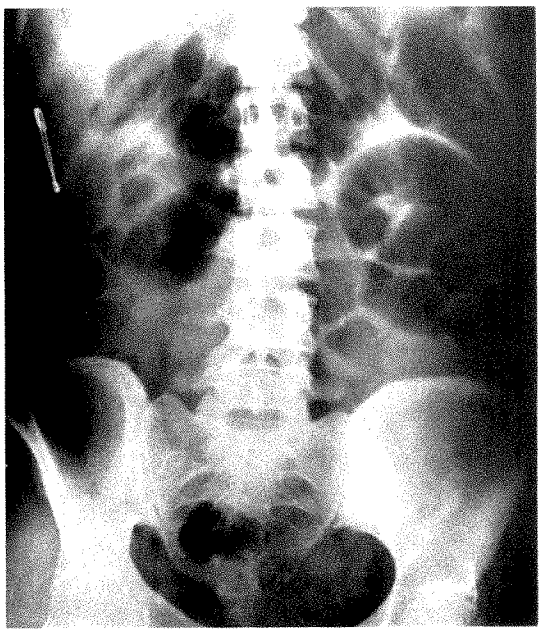


FIG. 6. Case II. Plain roentgenogram of the abdomen shows gaseous distention of the small intestine in the upper abdomen.

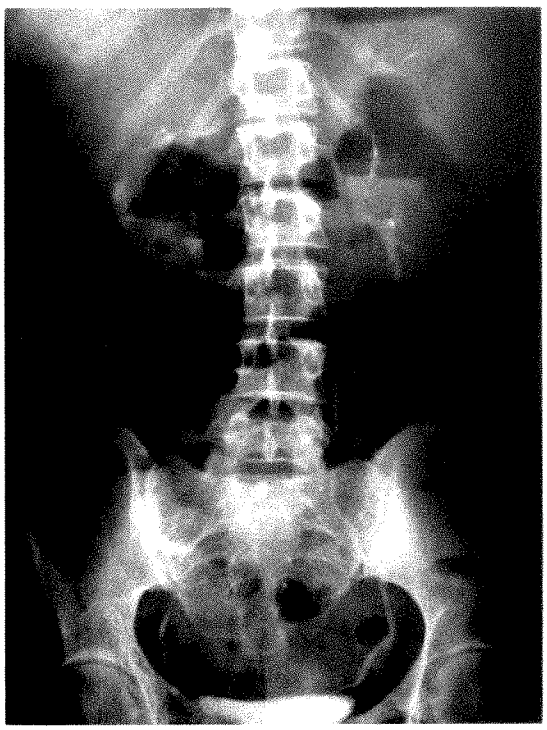


FIG. 7. Case II. Erect roentgenogram of the abdomen shows air fluid levels in the small intestine.

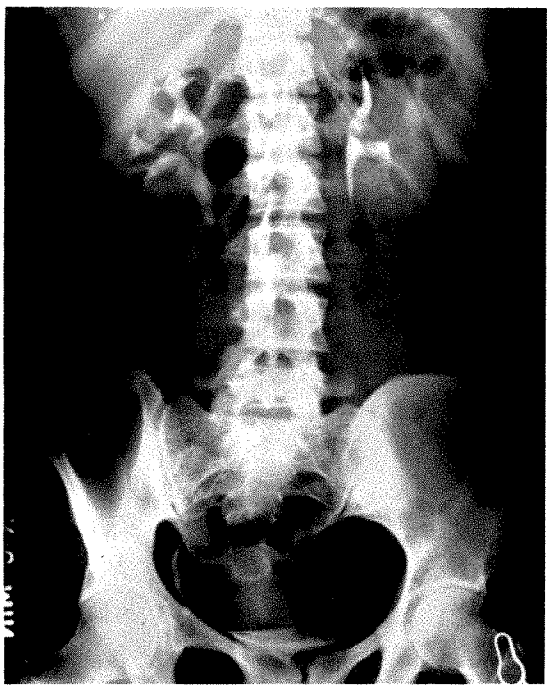


FIG. 8. Case II. Postoperative intravenous urogram shows that the right urinary tract has returned to normal. There is very little gas in the intestinal tract.

symptoms and the correct diagnosis was made because the presence of regional enteritis had previously been established. The 4 other cases reported in the literature were asymptomatic cases found by Redish[9] in a survey of 22 patients known to have regional enteritis.

In our first case, the diagnosis was not made until operation. Preoperatively, a diagnosis of neoplasm of the colon with retroperitoneal extension and ureteral obstruction was considered the most likely possibility. In our second case, the obstructed right ureter was noted at intravenous urography early in the study. During this same period, changing partial small bowel obstruction was noted on the abdominal roentgenograms. With our knowledge of the previous case two years before, the association of small intestinal obstruction and right ureteral obstruction suggested the preoperative diagnosis of regional ileitis. This was confirmed at laparotomy.

The partial ureteral obstruction in these 2 cases was due to edema associated with the terminal ileitis. The involved ileal segment in each instance was adherent to the retroperitoneal area with loose inflammatory adhesions. In Case 1 it was also adherent to the colon.

We believe that when a young adult is seen with partial right ureteral obstruction and partial small bowel obstruction the diagnosis of regional ileitis should be considered.

SUMMARY

Urinary tract involvement with regional enteritis is unusual. Partial ureteral obstruction in the absence of abscess formation is rare. Two cases are reported and added to those previously recorded in the literature.

We believe that, when a young adult is seen with roentgenographic evidence of partial right ureteral and small bowel obstruction, the diagnosis of regional ileitis should be considered.

C. Jules Rominger, M.D.
Misericordia Hospital
54th Street and Cedar Avenue
Philadelphia 43, Pennsylvania

REFERENCES

1. BRAASCH, W. F., and EMMETT, J. L. Clinical Urography. W. B. Saunders Co., Philadelphia, 1951.
2. BUCKSTEIN, J. The Digestive Tract in Roentgenology. Second edition. J. B. Lippincott Co., Philadelphia, 1953.
3. CROHN, B. B. Regional Ileitis. Grune & Stratton, Inc., New York, 1949.
4. GINZBURG, L., and OPPENHEIMER, G. D. Urological complications of regional ileitis. *J. Urol.*, 1948, *59*, 948–952.
5. GOLDEN, R. Roentgen-Ray Examination of the Digestive Tract. Thos. Nelson & Sons, New York, 1949.
6. HYAMS, J. A., WEINBERG, S. R., and ALLEY, J. L. Chronic ileitis with concomitant ureteritis, case report. *Am. J. Surg.*, 1943, *61*, 117–120.
7. LAPIDES, J. Personal communication.
8. NARATH, P. A. Renal Pelvis and Ureter. Grune & Stratton, Inc., New York, 1951.
9. REDISH, M. H. Ureteral complications and alterations of ureteral tonus in regional ileitis. *New England J. Med.*, 1952, *246*, 993–996.

MEDULLARY SPONGE KIDNEY*

By JOHN A. EVANS, M.D.
NEW YORK, NEW YORK

LINDVALL,[2] in a review of the literature and a report of 35 cases, discusses many aspects of medullary sponge kidney and, in particular, its roentgenologic features. On the basis of these 35 cases, Lindvall suggests that the disease is not as rare as the scant literature on the subject and the single case reports would indicate. Until this article was published, the roentgenographic appearance of this renal lesion was not generally appreciated. This, despite the fact that the clinical and roentgenologic features of the disease had been described by Lenarduzzi[1] in 1938. The term "medullary sponge kidney" was coined by Lenarduzzi to describe the appearance of the renal lesion.

The disease morphologically is an ectasia or cyst-like dilatation of the collecting ducts of the renal pyramids and hence is a lesion of the renal medulla (Fig. 1).

Recently, we have had the opportunity of seeing a patient (Case XI) with this renal lesion who died of unrelated disease. Autopsy examination permitted pathologic confirmation of the diagnosis. A review of our records revealed another autopsied case (Case XII) with the unequivocal gross and microscopic anatomic findings of medullary cystic disease of the kidneys. A third case (Case X) with the classic roentgen features of this lesion died in 1956 of unrelated disease. A review of the pathologic material available was unsuccessful in demonstrating the specific histopathologic findings of this disease, although at the time of the autopsy mention was made of some intratubular and interstitial calcification.

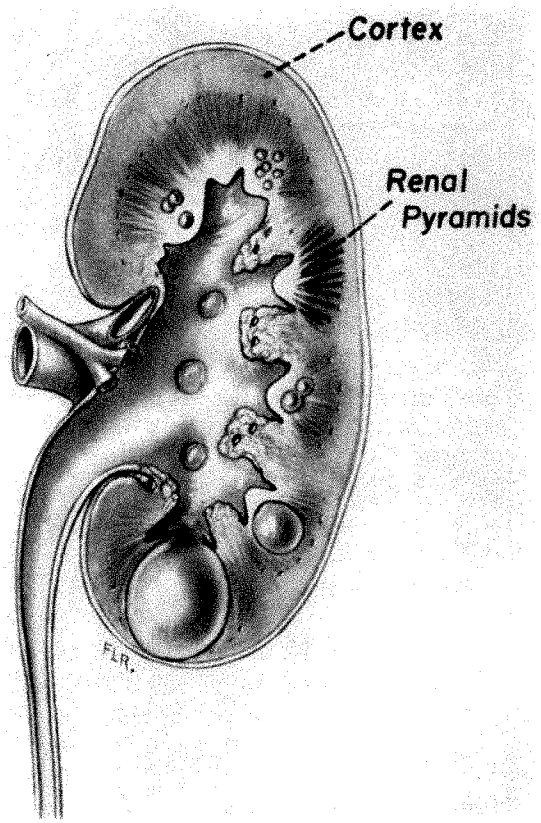


FIG. 1. Schematic drawing showing various stages of ectasia of the ducts of the renal pyramids. These range from minimal widening of ducts to formation of small cysts, then large cysts, many of which are shown connecting with calyces.

REPORT OF CASES

CASE I. L.G. (N.Y.H. #489999), a forty-five year old Negro male, had enjoyed good health until five months prior to hospital admission when he first noticed his urine to be dark amber with occasional brown flecks. There were no other urinary tract abnormalities and review of the systems was otherwise unremarkable. Physical examination revealed no abnormalities except for an elevated blood pressure of 195/120. It was stated that he was found to have had an elevated blood pressure on examination nine years previously.

Laboratory Findings. Urinalysis showed a faint trace of albumin, 2–4 red blood cells and 6–12 white blood cells per high power field.

* From the Department of Radiology, The New York Hospital—Cornell Medical Center.
Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

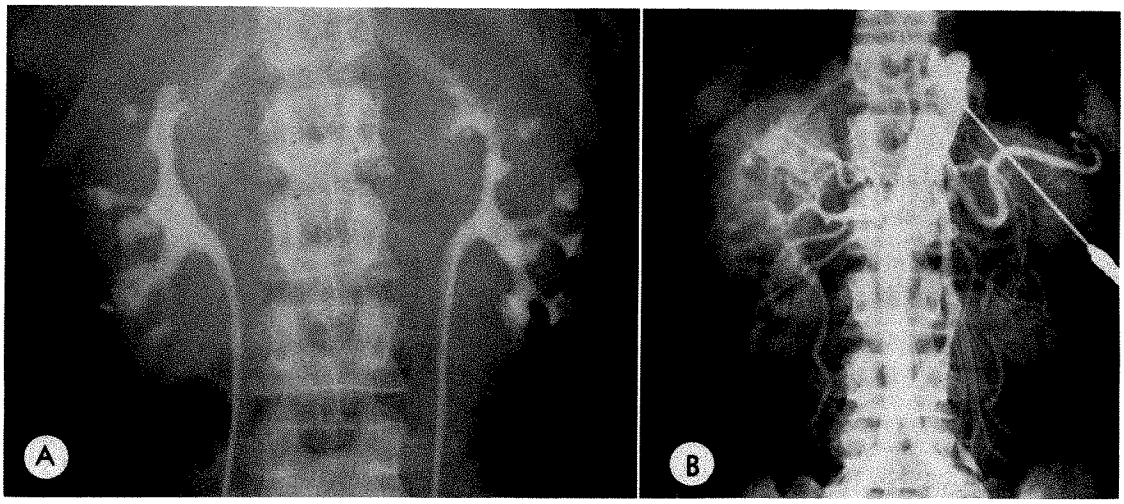


Fig. 2. Case I. (*A*) Retrograde pyelogram shows the small cysts and dilated collecting tubules communicating with the calyces in the renal pyramids. Urograms over the succeeding four years showed no change. The pyelographic image is highly characteristic of medullary sponge kidney. (*B*) Retrograde aortogram, second injection, reveals normal renal vasculature. Small discrete collections of contrast medium are seen in the cystic collecting tubules representing residua from the first injection.

Cultures were negative for acid fast bacilli. Papanicolaou stain was negative for malignant cells. Sickle cell preparations showed a 60 per cent sickling; blood chemistries were normal.

Roentgen Studies. Intravenous pyelography on October 17, 1956 revealed no opaque calculi on the preliminary scout roentgenogram. Following injection of opaque medium there was prompt bilateral excretion without any evidence of hydronephrosis. The striking finding, however, was the presence in both kidneys of minute collections of the opaque material within the collecting tubules of the renal papillae. In addition, some of the minor calyces were surrounded by an amorphous blush of the contrast material. A retrograde pyelogram on November 9, 1956 demonstrated the tubular ectasia and cysts to excellent advantage (Fig. 2*A*). The true nature of the lesion was not understood at this time and the roentgenologic diagnosis was acute necrotizing papillitis, although the patient's clinical course and subsequent roentgen examinations were not compatible with this diagnosis. A direct translumbar aortogram (Fig. 2*B*) demonstrated both renal arteries to be grossly normal.

Follow-up studies on this patient in 1957, 1958 and 1959 showed no essential change in the appearance of the kidneys on the interval urograms.

The patient has not developed demonstrable calculi. There have been no further episodes of hematuria. The blood pressure remains elevated but the patient has enjoyed good health.

Case II. R.T. (N.Y.H. #821215) a twenty-nine year old male, was seen by his doctor because of the recent occurrence of occipital headaches and a sense of tension. The headaches were of short duration and required no medication. The patient, otherwise, enjoyed good health. There were no urinary complaints. Physical examination revealed a normal healthy appearing adult whose examination disclosed no abnormalities except for a slightly elevated blood pressure of 136/98. Urinalysis and other routine laboratory studies were normal.

Roentgen Findings. An intravenous pyelogram demonstrated a large left kidney, no opaque calculi, prompt excretion and a startling collection of contrast agent in varying sized cyst-like cavities in the renal papillae of both kidneys (Fig. 3*A*). Many of the papillae were grossly enlarged, particularly throughout the left kidney. A retrograde study showed to good advantage filling of the various cysts (Fig. 3*B*). A delayed roentgenogram (Fig. 3*C*) revealed retention of the opaque medium in the cysts after emptying of the calyces. Nephrotomograms (Fig. 3, *D* and *E*) demonstrated in dramatic fashion the hyperplastic left kidney, the cysts and the large papillae.

Case III. J.C. (N.Y.H. #654606), a twenty-seven year old white female, was seen in our Out Patient Department for symptoms of acute cystitis, a condition for which she had been treated ten years previously. Physical examination revealed no significant abnormalities.

Laboratory Findings. Urinalysis showed a trace of albumin and an abundance of white blood cells. The other routine laboratory studies were normal

The patient responded quickly to appropriare therapy for her cystitis.

Roentgen Findings. During her workup, an intravenous pyelogram showed normally functioning, nonhydronephrotic kidneys. Both kidneys exhibited many striate densities in relation to the fornices of the minor calyces. In addition to the radiating densities, there were small cyst-like collections of the opaque material in a few papillae (Fig. 4, *A* and *B*). The examination was done without the use of ureteral compression.

Case IV. L.C. (N.Y.H. #761842), a forty-seven year old Negro male, entered the hospital because of hematuria and dysuria of two months' duration. Physical examination revealed no significant findings.

Cystoscopy revealed a tumor on the left an-

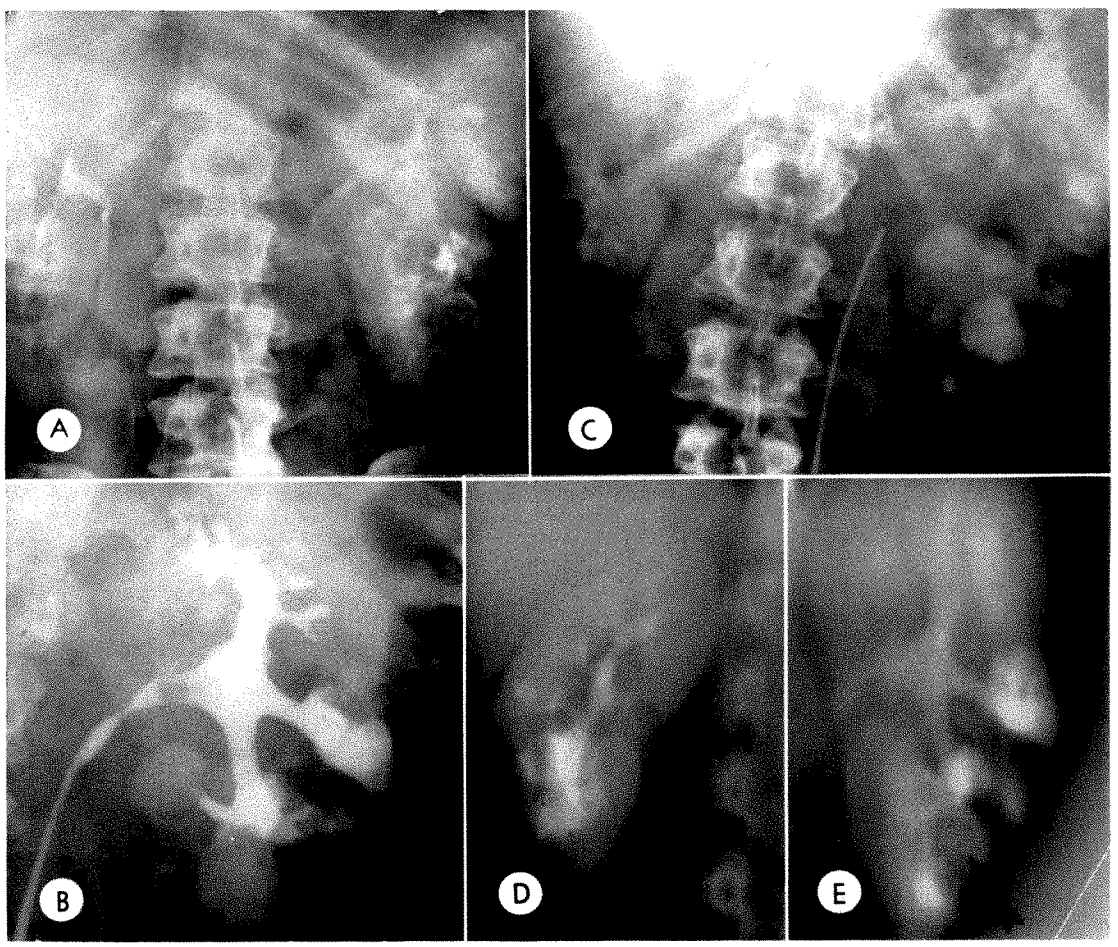


Fig. 3. Case II. (*A*) Intravenous pyelogram demonstrates bizarre collections of contrast medium in the ducts of the large hyperplastic renal pyramids. Beginning filling of large medullary cysts is noted. (*B*) Retrograde study shows the large left kidney and the presence of many large and small cysts of the renal pyramids connecting with the calyces. (*C*) Delayed roentgenogram after the retrograde study shows the retention of the contrast medium in the medullary cysts after most of it has been excreted from the collecting system. (*D* and *E*) Nephrotomograms of the right and left kidneys, with marked hyperplasia of the left. Multiple medullary cysts are seen in both kidneys. Large papillae (arrow) are also demonstrated.

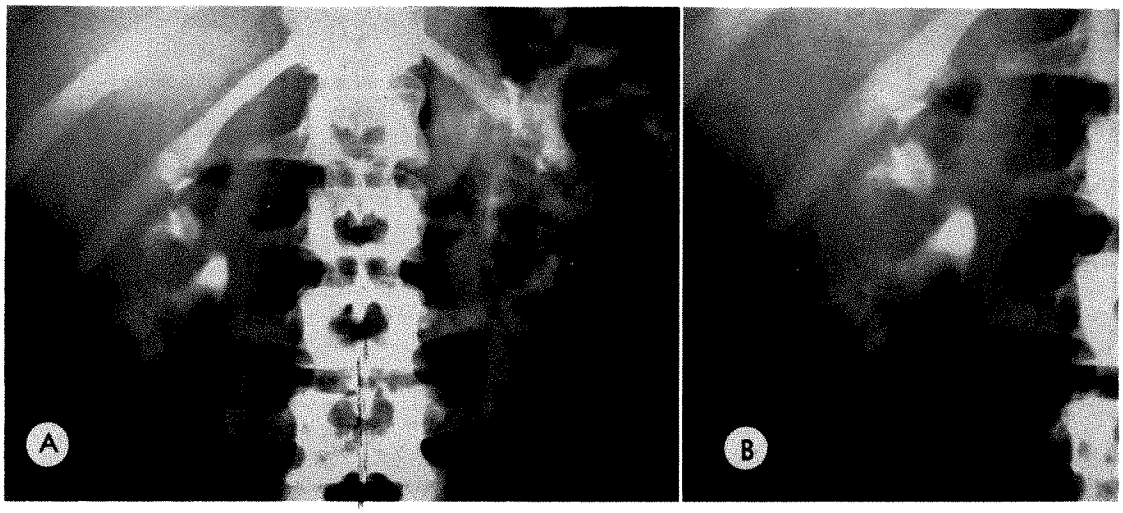


FIG. 4. Case III. (*A*) Intravenous pyelogram shows contrast medium in dilated collecting ducts of both kidneys, most pronounced on the right. (*B*) The spray-like appearance of the contrast medium in the collecting tubules of the right kidney is demonstrated.

terior wall of the bladder. There was a clear efflux from both ureters. Biopsy of the bladder tumor revealed carcinoma.

Laboratory Findings. Urinalysis showed gross and microscopic hematuria, a few white blood cells and traces of albumin. Repeated cultures and smears were negative for acid fast bacilli. Urine was also negative for heavy metals.

Roentgen Findings. An intravenous pyelogram revealed a few small punctate calcifications in the lower pole of the left kidney. The postinjection roentgenograms demonstrated prompt bilateral excretion. Surrounding each of the minor calyces were small collections of contrast agent having both a striate and nodular appearance. These were located in the papillae and at the time of this examination suggested necrotizing papillitis. There were no other symptoms to support this diagnosis and subsequent roentgen studies showed no progression or change in the appearance of the kidneys. The retrospective appearance is quite typical of what Lindvall has described as medullary sponge kidney (Fig. 5, *A* and *B*).

The patient, despite aggressive surgery and radiation therapy, expired from his disease approximately five months after being first seen. Autopsy permission was not granted.

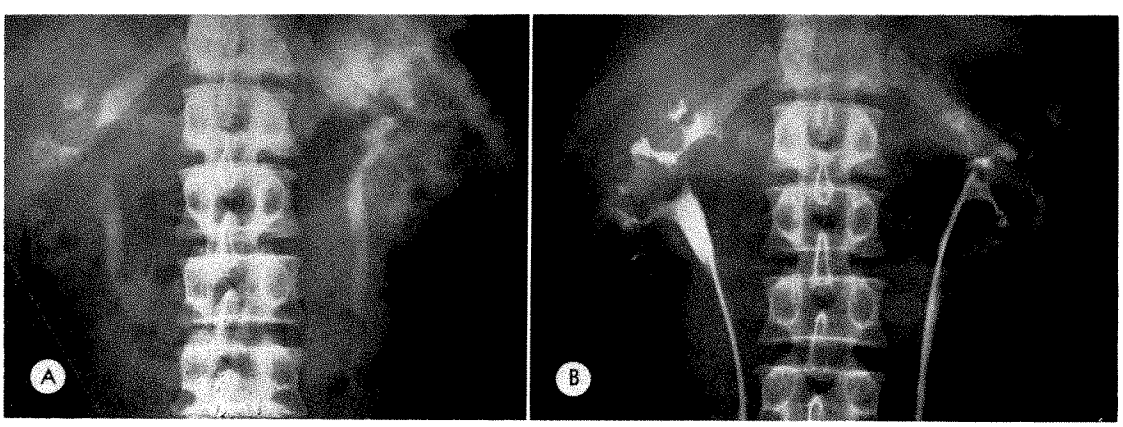


FIG. 5. Case IV. (*A*) Intravenous pyelogram shows the minor calyces to appear fuzzy and indistinct because of collections of the contrast agent in the collecting tubules of the renal papillae. This appearance is very similar to the early stage of necrotizing papillitis. (*B*) Retrograde study shows filling of dilated, ectatic, cystic collecting tubules of the renal pyramids.

Case v. A.F. (N.Y.H. #753366), a thirty-seven year old nurse, had intravenous pyelography because of slight elevation of diastolic pressure noted on a pre-employment examination. Her blood pressure was 130/90; physical examination showed no significant findings.

Laboratory Findings. Urinalysis and other routine laboratory studies were normal.

Roentgen Findings. An intravenous pyelogram revealed a small ring-like calcified density over the upper pole of the right kidney. The postinjection studies localized this in one of the minor calyces of the upper pole. In addition, striate collections of the opaque material were seen in the papillae of the calyces in the lower pole of this kidney, representing contrast agent in the collecting ducts of the papillae. The left kidney demonstrated a small cyst-like collection of the opaque medium adjacent to one of the minor calyces of the middle calyceal group. A few of the papillae of the right kidney were somewhat enlarged. Ureteral pressure was not used. The findings of calcification, visualization of collecting ducts, somewhat enlarged papillae and a cyst-like collection in the left kidney are sufficient to warrant the roentgenologic diagnosis of medullary sponge kidney (Fig. 6).

Case vi. B.R. (N. Y. H. #745315), a forty-four year old female, was admitted to the hospital for surgery of a carcinoma of the breast. The pertinent past history revealed that three–four years prior to admittance she had dysuria

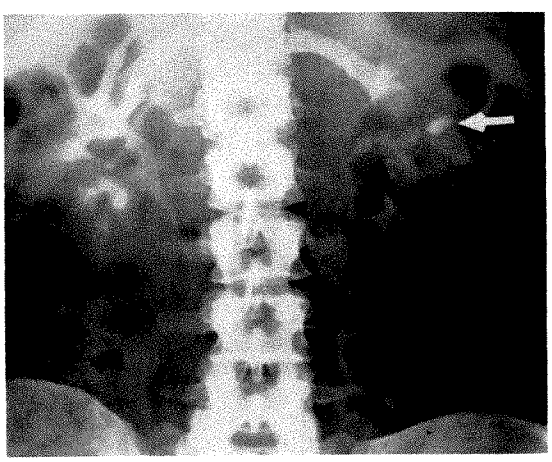


Fig. 6. Case v. Intravenous pyelogram demonstrates tubular ectasia producing spray-like, striate densities in the renal pyramids. The minor calyces are somewhat exaggerated, reflecting slight enlargement of the renal pyramids. There is a clearly defined medullary cyst in the left kidney (arrow).

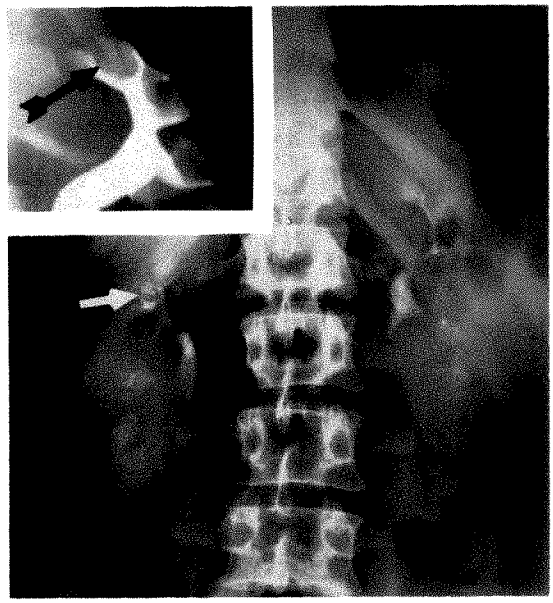


Fig. 7. Case vi. Intravenous pyelogram demonstrates opaque medium in the dilated collecting tubules, producing radiating linear densities extending from the minor calyces. Some of the minor calyces, due to enlargement of the papillae and nonfilling, have what Lindvall has referred to as a "blistered" look (white arrow). Inset demonstrates the large deeply cupped minor calyces due to enlargement of papillae.

with several episodes of hematuria and concurrent suprapubic discomfort before voiding. Cystoscopy at that time disclosed "polyps." At the time of her present admittance she had stress incontinence, frequency and incomplete voiding.

Laboratory Findings. Urinalysis was negative; serum calcium and phosphorus were normal.

Roentgen Findings. An intravenous pyelogram showed prompt function of nonhydronephrotic kidneys. The contrast medium was seen in the collecting tubules of the papillae of both kidneys. Some of the papillae had a clear ring-like appearance that Lindvall has referred to as a "blistered look." A retrograde study showed no reflux of the contrast material into the collecting tubules but did show the papillae to be somewhat large. This was demonstrated by the rather deeply cupped appearance of the minor calyces (Fig. 7).

Case vii. I.A. (N. Y. H. #816719), a twenty-nine year old Negro woman, was found to have a slight elevation of blood pressure during

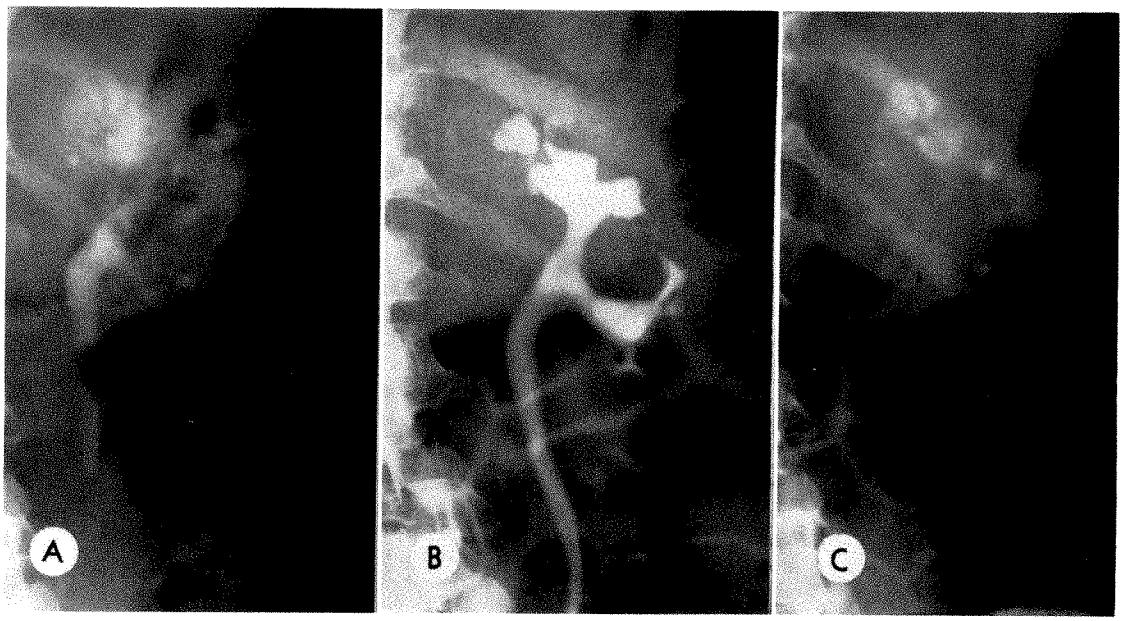


FIG. 8. Case VII. (*A*) Intravenous pyelogram shows the contrast medium in the large cysts of the renal pyramids of the upper pole of the left kidney. Many small cysts connecting with calyces are seen. (*B*) Retrograde study of the left kidney shows filling of only some of the cysts, one of which looks like a calycine diverticulum. (*C*) Delayed roentgenogram shows retention of the opaque medium in the cysts and emptying of the calyces and renal pelvis.

pregnancy. During the course of a workup to evaluate the blood pressure, roentgenograms were made of the upper urinary tracts.

Laboratory Findings. Urinalysis was negative for red blood cells, white blood cells, albumin or casts. Cultures were negative for acid fast bacilli. Blood chemistries were normal.

Roentgen Findings. An intravenous pyelogram revealed deposits of contrast medium in many of the papillae of the left kidney, arranged in radiating strand-like densities and smudgy amorphous collections surrounding the minor calyces. Retrograde pyelograms showed many small cysts in the pyramids of the left kidney (Fig. 8, *A*, *B* and *C*). An examination six months later demonstrated the same pyelographic findings now considered to be characteristic for medullary sponge kidney.

CASE VIII. A.M. (N.Y.H. #797519), a twenty-three year old white female, is a well documented case of lupus erythematosus. During the course of several hospital admissions she was found at roentgenography to have bilateral nephrocalcinosis.

Laboratory Findings. Urinalysis showed occasional red blood cells, 8–12 white blood cells, and a few granular casts. The sedimentation rate was 9 mm. Blood chemistries showed the blood urea nitrogen, sugar, urea clearance, phenolsulfonphthalein and Remond-Smith test to be normal.

Roentgen Findings. A review of the intravenous pyelograms revealed normal bilateral function and no hydronephrosis. Upon close inspection, however, in addition to calcific deposits in the renal pyramids, dilatation of the collecting tubules was noted (Fig. 9).

CASE IX. P.F. (N.Y.H. #705577), a fifty-four year old white male, had a long history of renal disease marked by periodic exacerbations of hematuria and pain. The patient has had thirteen hospital admissions and has been treated for gouty nephropathy, chronic pyelonephritis and hypertension.

Intravenous pyelograms over this long period of observation have shown evidence of chronic pyelonephritis. In addition to these changes, there were seen small discrete cyst-like collections of the contrast medium as well as some smudgy collections in somewhat enlarged papillae (Fig. 10). Initially, it was thought that these changes represented necrotizing papillitis or possibly renal tuberculosis. Neither diagnosis, however, was supported by the clinical

course or laboratory findings. Cultures have been repeatedly negative for acid fast bacilli. It is now felt that the urographic findings are consistent with renal papillary necrosis as well as medullary sponge kidney.

Case x. M.S. (N.Y.H. #334337), a seventy-three year old female, had Laennec's cirrhosis and bleeding esophageal varices. A splenoportal venogram demonstrated obstruction of the portal vein and a splenorenal shunt was performed.

She did poorly postoperatively and died seven days after the operation. During her hospital workup, intravenous and retrograde pyelograms were made. The retrograde pyelogram demonstrated the presence of many small cyst-like cavities in the papillae of the renal pyramids. In addition contrast medium was seen in the dilated collecting tubules of the renal pyramids. The roentgen appearance was typical for that of medullary sponge kidney (Fig. 11).

Autopsy revealed carcinoma of the liver with invasion of the portal vein. There was metastatic tumor in the lungs and esophageal and gastric varices. The kidneys showed a number of infarcts. The areas of the cortex showed extensive atrophy of the tubules, fibrosis of glomeruli, intimal thickening of the small arteries and interstitial infiltration with lymphocytes. In the pyramids there was intratubular and interstitial calcification.

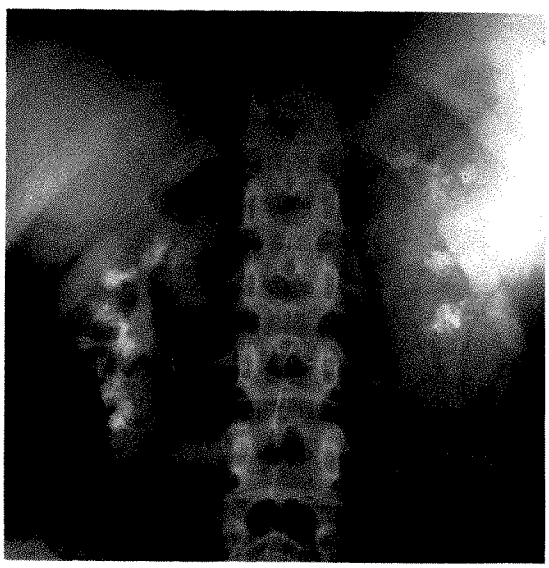


Fig. 9. Case viii. Intravenous pyelogram shows nephrocalcinosis with small calcific deposits in the collecting ducts of the pyramids. The opaque medium is seen in the dilated, ectatic ducts.



Fig. 10. Case ix. Pyelogram shows the many small communicating collections of contrast medium in the medullary portion of the kidney along with the somewhat enlarged pyramids. These findings are highly suggestive of sponge kidney and renal papillary necrosis.

Case xi. H.N. (N.Y.H. #836571), a fifty-six year old male, was admitted to the hospital because of severe hypertension which had been first noted five years prior to admission. Two months prior to admission he noted onset of excessive fatigability, frequency and nocturia, burning, urgency and ankle edema. Examination revealed an acutely ill white male with twitching in all extremities and profuse diaphoresis. An eye examination showed early papilledema, old and new exudates, flame shaped and round hemorrhages and extreme spasm of the retinal arterioles. The neck veins were distended, the heart was enlarged to the left and there was a blowing apical systolic

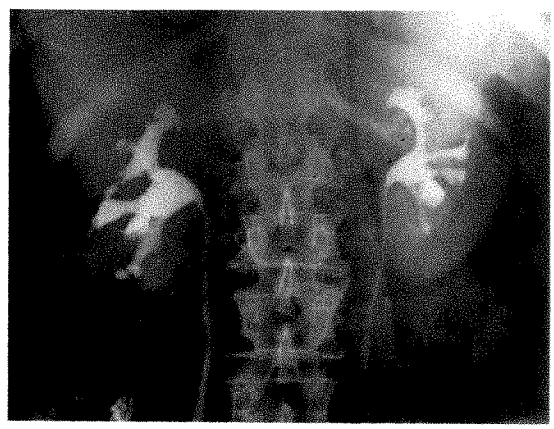


Fig. 11. Case x. Retrograde pyelogram demonstrates multiple small cysts in the pyramids, presenting the appearance of diverticula.

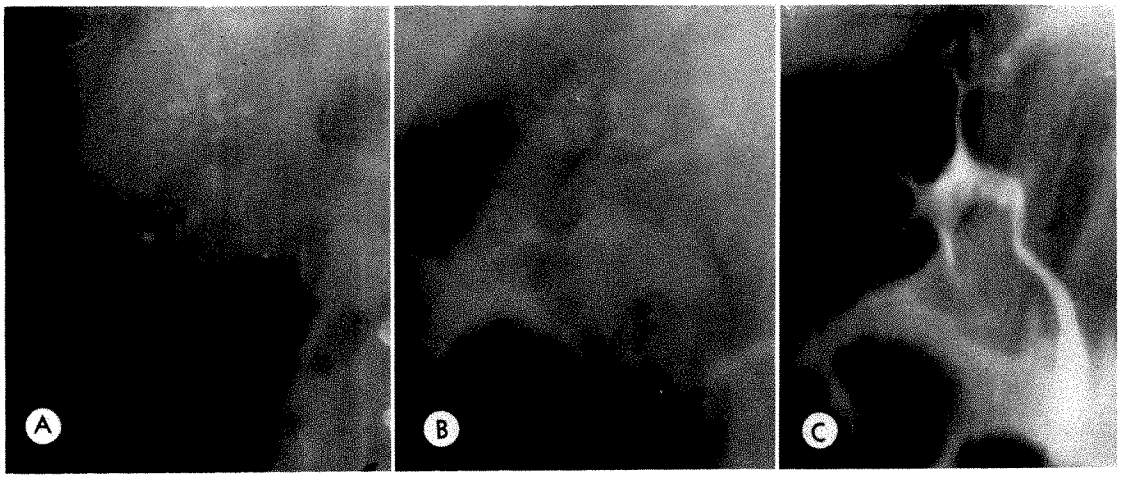


FIG. 12. Case XI. (*A*) Roentgenogram shows multiple small clusters of calcified deposits. The size, arrangement and grouping are highly suggestive of cystic lesions of the renal pyramids. (*B* and *C*) Intravenous pyelograms reveal dilated ducts and cysts of the renal pyramids of the upper pole of the kidney.

murmur. The liver was enlarged downward 4 finger breadths. His blood pressure was 200/140.

Laboratory Findings. Urinalysis showed 4 plus albumin, 10–20 red blood cells, 15–20 white blood cells per high power field and many casts. Cultures were negative for acid fast bacilli. The complete blood cell count was hematocrit 30 and white blood cells 16,600. The blood urea nitrogen, on admission and subsequently, fluctuated between 126 and 24. The electrocardiogram showed auricular fibrillation and evidence of coronary artery disease. Blood chemistries revealed evidence of hypokalemic alkalosis. The patient responded poorly to therapy for his malignant hypertension and renal failure and expired two months following his hospital admission.

Roentgen Findings. Roentgenograms of the upper urinary tracts revealed multiple small discrete calculi in the right kidney (Fig. 12*A*). Intravenous pyelograms revealed ectasia and small cyst formation of the ducts of the renal papillae in the upper pole of the right kidney. The calculi were located in the ectatic, dilated ducts (Fig. 12, *B* and *C*).

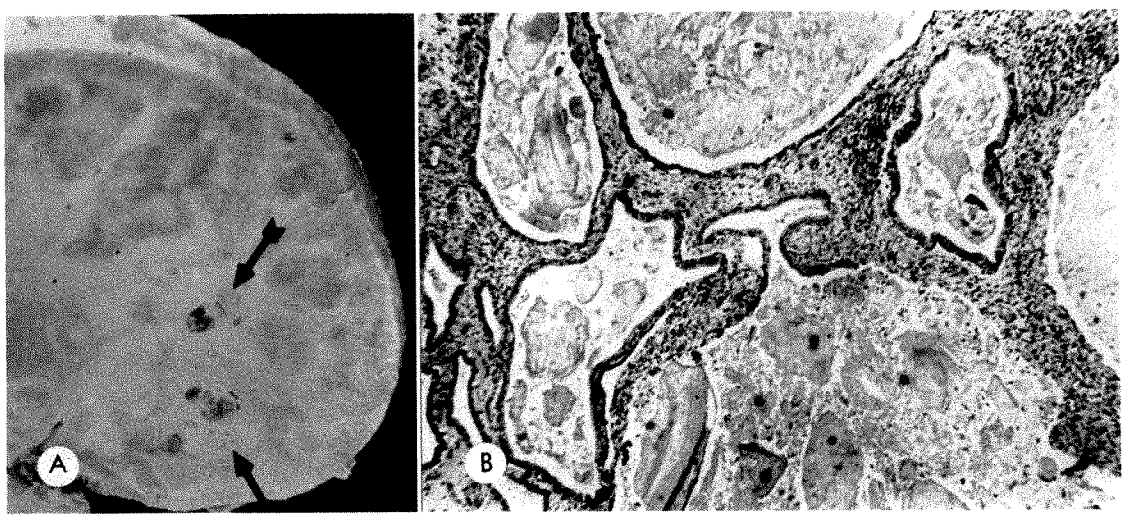


FIG. 13. Case XI. (*A*) Gross autopsy specimen shows the presence of many cysts in the pyramids of the upper pole of the right kidney (arrows). (*B*) Microscopic section shows dilated, ectatic ducts lined with low cuboidal epithelium.

At autopsy, the pertinent findings to this report were those confined to the kidneys. These revealed arterial and arteriolar nephrosclerosis; both kidneys weighed 290 gm.; finely granular cortical surfaces and uniformly thinned cortices were found. There was marked narrowing of the lumina and thickening of the walls of the arteries and arterioles due to fibrous tissue, hypertrophy of the muscularis and proliferation of the cells in the intima. The cortex of the right kidney contained thin walled cysts filled with clear fluid and measuring up to 3.5 cm. in diameter, and similar cysts, measuring up to 0.5 cm. were situated in the medulla (Fig. 13*A*). The three superior calyces of the right kidney and corresponding papillae contained brown, friable calculi. Microscopic examination demonstrated marked cystic dilatation of the medullary tubules. The cysts were lined by a single layer of low cuboidal epithelium (Fig. 13*B*).

Case xii. M.L. (N.Y.H. #339240), an eight year old girl, was admitted to The New York Hospital for the first time on September 12, 1942 because of intractable anemia. Between that date and the day of death, May 22, 1943, there were six hospital admissions. Death occurred from renal insufficiency and the anemia was directly related to the renal lesion.

At autopsy, gross examination revealed innumerable cysts sharply limited to the renal pyramids. The majority of the cysts were from 1–5 mm. in diameter. The cysts were thin walled and contained clear, colorless, odorless fluid. Microscopic examination of the renal pyramids revealed numerous thin-walled cysts among the loops of Henle and the collecting tubules, many of which were not visible grossly. There was no inflammation near the cysts. The cysts were lined by a layer of low cuboidal cells which closely resembled the cells of the collecting tubules (Fig. 14).

(This case has been reported previously.[4])

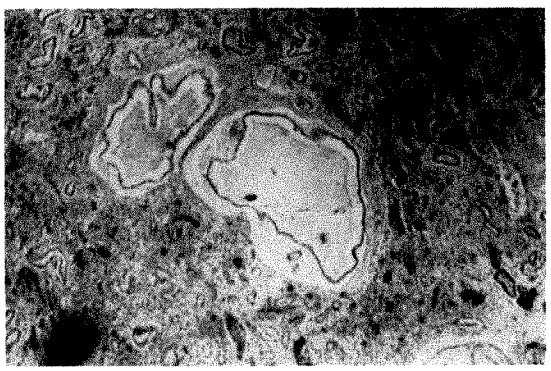


Fig. 14. Case xii. Microscopic section shows dilated, cystic, collecting tubules lined by a single layer of low cuboidal epithelium.

Discussion

The etiology of medullary sponge kidney is not completely understood. In all probability it represents a congenital defect in development of the collecting tubules in the renal medulla. The dilatation of the ducts of the renal pyramids and papillae may vary from slight widening to the formation of large cyst-like cavities. The extent of the disease also varies from involvement of one or several papillae of one kidney to diffuse bilateral involvement. The affected papillae may enlarge and result in general enlargement of the kidney.

Minimal enlargement of the collecting ducts will, on excretory urograms, be seen as spray-like densities radiating from a minor calyx (Fig. 4*B*). The appearance is similar to, but somewhat more pronounced than, that of simple stasis within the ducts. Differentiation in such cases may be possible only if there is enlargement of the involved renal pyramids. Visualization of the renal papilla as a physiologic occurrence is always a transient phenomenon, whereas visualization of the ectatic collecting ducts of medullary sponge kidney is constant and reproducible. Extreme dilatation of the ducts results in the formation of cyst-like cavities, usually connecting with the minor calyces. The roentgenographic appearance of the advanced form of the disease produces a startling bizzare picture of ectatic tubules, multiple cysts, large papillae and occasionally large kidneys (Fig. 3, *A–E*). The correct diagnosis in such instances can be made by the specific and characteristic pattern seen on the pyelogram.

The cysts are best demonstrated by excretory urography where they fill with the contrast medium ahead of the pyelocalyc-

eal system and retain the contrast agent after the calyceal system has been emptied.

Calculi, usually small and discrete, are frequently seen in the cyst-like cavities. They were present in all but 2 of Lindvall's cases and in 5 of our cases. The presence of calculi tends to provoke such complications as obstruction, hematuria and infection.

The disease is said to be confined to the medulla. In one of our autopsied cases, several cysts were also seen in the cortex and probably represented simple retention cysts.

Reilly and Neuhauser[3] recently reported 11 cases of renal tubular ectasia in infants and children with a high association of cystic disease of the liver and pancreas. The morphologic character of the renal lesions in our cases is similar to that reported by these authors, but in none of our cases was there anatomic or clinical evidence of associated cystic disease of the liver or pancreas.

The major roentgenologic considerations in the differential diagnosis are renal tuberculosis, hyperparathyroidism, necrotizing papillitis and calyceal diverticula. None of these diseases should provide serious difficulty. Necrotizing papillitis in our experience has been a fatal disease. Extensive necrosis of the renal papillae presents large ragged excavations on pyelograms. It is also usually associated with some degree of obstructive uropathy or diabetes. Nephrocalcinosis associated with hyperparathyroidism may offer some difficulty but the other stigmata of the disease are usually sufficient to differentiate it from medullary sponge kidney. The ragged cavitary defects of renal tuberculosis are not limited to the papillae; also other evidence of the disease is usually adequate to differentiate it from sponge kidney.

SUMMARY

Twelve cases possessing the roentgenographic and/or pathologic criteria of medullary sponge kidney have been presented. The anatomic and roentgenologic features of the disease have been discussed and illustrated.

525 East 68th Street
New York 21, New York

REFERENCES

1. Lenarduzzi, G. Reperto pielografico pocco commune (dilatazione delle vie urinarie intrarenali). *Radiol. med.*, 1939, *26*, 346.
2. Lindvall, N. Roentgenologic diagnosis of medullary sponge kidney. *Acta radiol.*, 1959, *51*, 193–206.
3. Reilly, B. J., and Neuhauser, E. B. D. Renal tubular ectasia in cystic disease of kidneys and liver. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 546–554.
4. Smith, C. H., and Graham, J. B. Congenital medullary cysts of kidneys with severe refractory anemia. *Am. J. Dis. Child.*, 1945, *69*, 369–377.

VOL. 86, NO. 1

THE ROENTGEN MANIFESTATIONS OF BILATERAL RENAL CORTICAL NECROSIS*

By WILLIAM H. McALISTER, M.D.,† *and* STANLEY H. NEDELMAN, M.D.
CINCINNATI, OHIO

BILATERAL renal cortical necrosis as a cause of acute renal failure was first recognized by Juhel-Renoy in 1886.[20] By definition, this is a condition characterized by bilateral symmetric ischemic necrosis involving the cortical portions of the kidneys, including the columns of Bertini, with relative sparing of both the medulla and a rim of subcapsular cortical tissue.[13] In the past, little attention has been directed to the roentgen aspects of this disorder since its rapidly fatal course was unassociated with specific roentgenographic features.

In recent years patients with bilateral renal cortical necrosis have survived for longer periods of time due to a better understanding of this disorder and to the availability of improved supportive measures in the treatment of acute renal failure. In some instances this prolonged survival has afforded sufficient time for the mineralization of necrotic cortical tissue, an alteration detectable by roentgen means.

Although Wells, Margolin and Gall[20] state that less than 160 cases of renal cortical necrosis have been reported through 1959, undoubtedly many more cases have gone unrecognized or unreported. This figure does not include the 21 cases described by these authors. A review of the literature has disclosed 4 cases which exhibited antemortem roentgenographic findings characteristic of renal cortical necrosis.[2,6,11,15] In only one article was specific attention called to the roentgen features.[15] The present paper deals with a case of bilateral renal cortical necrosis in which, following survival of the acute episode, cortical calcification as well as shrinkage of the kidneys developed.

REPORT OF A CASE

A twenty-four year old white female was transferred from another hospital for management of acute renal insufficiency. She had been in her sixth month of pregnancy when nausea, vomiting, and abdominal cramps appeared. Her blood pressure at that time was 160/100, and she was considered to be in a pre-eclamptic state. Severe vaginal bleeding promptly followed and a stillborn fetus was delivered by emergency low cervical cesarean section. The clinical impression of placenta previa was substantiated. Considerable blood loss occurred prior to and during the operation, although no blood pressure reading below 120/80 was recorded. Four units of blood, properly cross-matched, were administered without reaction during the operation and within forty-eight hours of its termination. As a result of these transfusions, the hemoglobin rose from 4 to 10.5 gm./100 ml.

Oliguria developed immediately following the operation. Over the next five days the urine output varied between 25 and 50 cc. per day. The blood urea nitrogen rose to 135 mg./100 ml. and remained between 100 and 200 mg./100 ml. for the next thirty-five days. On the fourth postpartum day, at the time of transfer to the Cincinnati General Hospital, pulmonary congestion, peripheral edema, and abdominal distention were present. Over the next three days the serum potassium rose to 7.9 mEq./liter, and there was electrocardiographic evidence of hyperkalemia. Following hemodialysis with an artificial kidney there was considerable improvement in the blood chemistry values.

Bilateral flank pain, which appeared early in the course of her disease, subsided and the urine, which had been grossly bloody, cleared. The urinary albumin ranged from 2 to 4 plus. Antibiotic agents were administered for pyuria and bacilluria which developed as a result of an indwelling catheter which had previously been inserted.

Three days after hemodialysis acute pulmonary edema developed and her clinical condition deteriorated. Hemodialysis was repeated, and again there was clinical improvement. A

* From the Department of Radiology, University of Cincinnati College of Medicine, and the Cincinnati General Hospital.
† Trainee, National Cancer Institute.

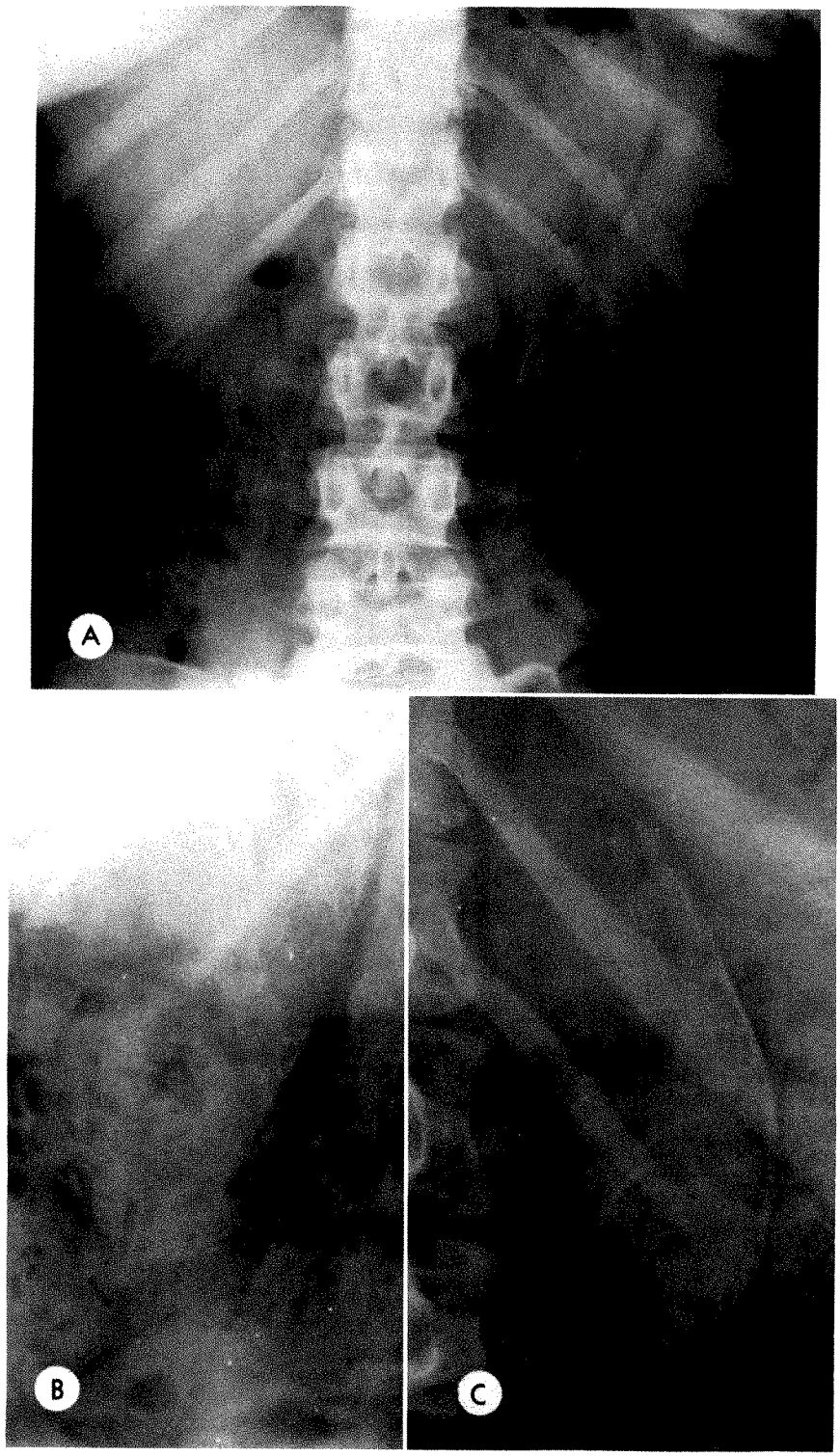


FIG. 1. (*A*) Plain roentgenogram of the abdomen taken on the fifty-seventh day of illness. A narrow rim of renal cortical calcification is seen bilaterally. The obvious splenomegaly is an incidental finding. (*B* and *C*) Oblique roentgenograms of each kidney demonstrate the cortical calcification to better advantage.

few days later pulmonary edema recurred. This was accompanied by hemorrhage from the vagina and from the site of a tracheostomy. Dehiscence of the abdominal incision occurred and was closed with wire sutures. Loud pleural and pericardial friction rubs were audible and there was cardiac arrhythmia. The mental confusion and delirium which followed the first hemodialysis did not improve.

Three weeks after the first dialysis the urinary output, which had ranged between 25 and 200 cc. per day, rose to normal levels while the blood urea nitrogen remained at about 200 mg./100 ml.; three weeks later it had fallen to 84 mg./100 ml. Anemia and splenomegaly (the latter was first noted several days following cesarean section) persisted. The anemia was attributed to chronic blood loss. Hypersplenism was not felt to be present.

On the fifty-seventh day following delivery, abdominal roentgenograms were made preliminary to a renal biopsy (Fig. 1, *A*, *B* and *C*). These revealed moderately extensive renal cortical calcifications. These were more apparent on laminagrams (Fig. 2, *A* and *B*). It was also apparent that both kidneys had decreased considerably in size when compared with roentgenograms two months earlier. Over the ensuing nineteen months the albuminuria and mild hypertension have persisted. Several bouts of urinary tract infection have been treated successfully. The blood urea nitrogen and creatinine have fallen to normal levels although low urea and creatinine clearances have been obtained repeatedly. During this period there has been a slight increase in the extent of cortical calcification and a further slight reduction in the size of the left kidney. A recent intravenous pyelogram (Fig. 3, *A* and *B*) demonstrated prompt excretion with moderate concentration of the contrast material. Normal pelvicalyceal systems were observed bilaterally.

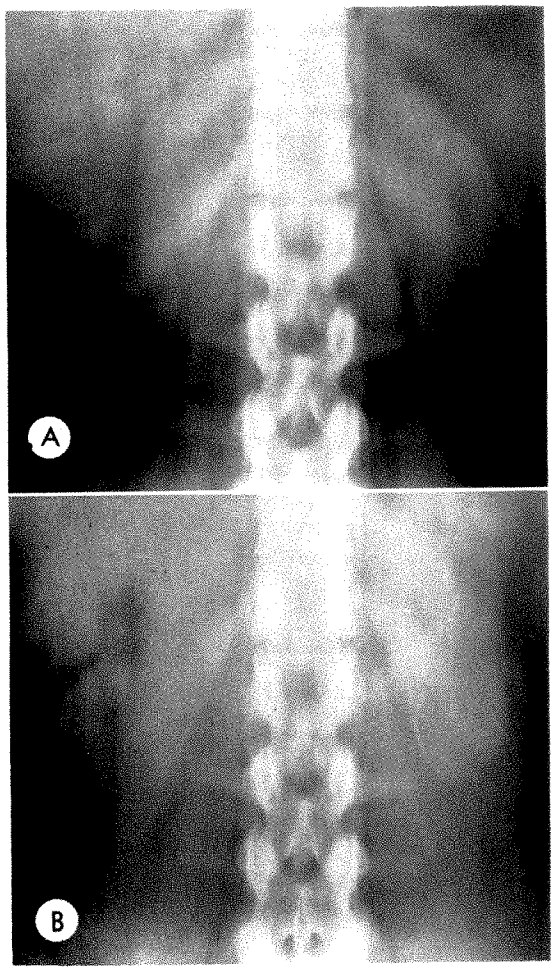


FIG. 2. (*A*) Laminagram demonstrates the calcification well and shows its cortical distribution. (*B*) Laminagram seventeen months later. There is a slight increase in the amount of calcification and a further slight decrease in the left renal size.

DISCUSSION

Bilateral renal cortical necrosis has long been recognized as a cause of acute renal failure. A great variety of apparently unrelated conditions have been observed to antedate the abrupt onset of this disorder. It has occurred in association with severe burns, multiple fractures, internal hemorrhage, major surgical procedures, severe infections, malignant neoplasms, transfusion of incompatible blood, the administration of a variety of nephrotoxins, favism, diarrhea with dehydration, influenza, perforated peptic ulcer, peritonitis, and periarteritis nodosa. The occurrence in pregnancy (usually in association with abruptio placentae)[1,13] has been frequently emphasized in the literature. Duff and More[7] collected 71 examples of cortical necrosis through 1941 and noted that 67 per cent were associated with pregnancy. Wahle and Muirhead[19] also reviewed the literature on this subject through 1953 and observed that 57 per cent of 149 cases were associated with pregnancy. A recently published series of 21 cases reported by Wells, Margolin,

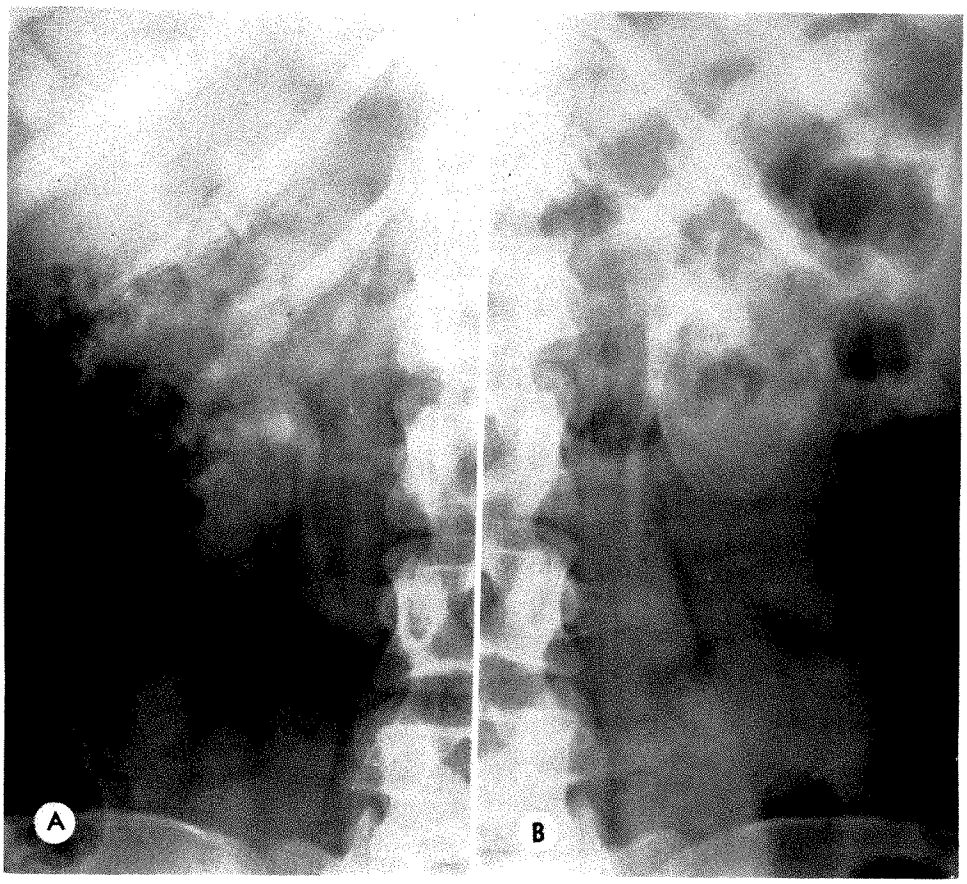


FIG. 3. (*A* and *B*) Intravenous pyelograms one and one-half years after the acute episode show prompt excretion bilaterally with only moderate concentration of the contrast material. The pelvicalyceal systems appear normal.

and Gall[20] is at variance with the surveys cited, in that only 14 per cent of their cases were preceded by the hemorrhagic complications of late pregnancy. No definite conclusion can be drawn from this relatively small group of cases. The more frequent use of needle biopsy in suspected cases of cortical necrosis may substantiate their implication that the high degree of association with pregnancy is more apparent than real.

No specific etiologic agent has been uncovered as the cause of cortical necrosis. However, renal ischemia is considered the pathogenetic factor of greatest importance in the production of the characteristic necrotic cortical lesions. The mechanism of ischemia, though extensively investigated, has not been satisfactorily explained. The theory of renal vasospasm has received the widest support. Davis feels that cortical necrosis is produced by transitory vasospasm of intrarenal arteries for periods of from perhaps two to four hours, followed by the restoration of blood flow.[5] No vasopressor substance has as yet been isolated from patients with cortical necrosis. The experimental production of vasospasm sufficient to cause cortical necrosis has centered about toxins, poisons, and vasoconstrictor substances.[13] Included among these are serotonin and mercuric chloride. Other investigators have implicated embolism, vascular shunts, vasoparalysis, vascular damage, and fibrin deposition as causative factors in the induction of renal ischemia.[7,8,13,18]

Although the entire renal cortex may be-

come necrotic, Duff and More[7] state that usually only its outer one-half to two-thirds is affected. A narrow rim of normal subcapsular parenchyma is often spared, presumably by reason of collateral capsular circulation. If the patient survives cortical necrosis, the recovery of renal function is more or less incomplete, in contradistinction to the complete restoration of function in acute tubular necrosis.[10] The outcome will depend to a considerable extent on the amount of tissue destroyed.[20] Even with extensive cortical necrosis, prolongation of life through meticulous care to the problems of fluid and electrolyte balance as well as by the use of hemodialysis has allowed sufficient time for the calcification of necrotic cortical tissue, as in the present case.

As stated earlier, a review of the literature disclosed 4 cases of renal cortical necrosis in which cortical calcification has been demonstrated roentgenographically in the living patient. The calcifications were first observed at seven weeks,[15] fifty-seven days,[6] two months,[2] and sixty-one days[11] after the onset of the acute episode. In our own case the cortical calcification was first seen on the fifty-seventh day. In DeGraeff and DeBaan's[6] case microscopic calcification was observed on a renal biopsy taken on the twentieth day and cortical calcification was seen on the plain roentgenograms of the abdomen on the fifty-seventh day.

In a case reported by Gjørup, Killmann, and Thaysen,[10] diffuse cortical calcification was found on a renal biopsy specimen obtained on the thirty-eighth day and a thick renal cortical calcific shell was observed at the time of necropsy on the fifty-first day. No roentgenograms had been obtained. According to Sheehan and Moore,[18] as well as other investigators, calcification in this condition is an uncommon finding at the time of autopsy. A review of 21 cases encountered at the Cincinnati General Hospital[20] failed to demonstrate renal calcification at necropsy. However, death occurred in these cases, as in a majority of those reported, within five to thirty-one days.

From the information available it appears that a prerequisite to the roentgenographic demonstration of cortical calcification in cortical necrosis is survival of the patient for at least one and one-half to two months. Mortensen *et al.*[16] have pointed out that there is not always a close correlation between the quantity of calcification visible microscopically and that demonstrated roentgenographically. In some instances the calcification demonstrated microscopically may not be visible on roentgenograms. Moëll[15] reported a case in which cortical calcification was seen on a postmortem roentgenogram of the kidneys but not on roentgen studies made prior to death.

DIAGNOSIS

The diagnosis of bilateral renal cortical necrosis is definitively made by biopsy or at the autopsy table.[4,17] Some investigators feel that there are clinical features of practical value in differentiating this type of acute renal failure from that occurring in acute tubular necrosis, acute glomerulonephritis, acute bilateral renal arterial or venous occlusion, and extensive bilateral renal infarction.[20] The frequent association of cortical necrosis with the hemorrhagic complications of late pregnancy is emphasized, as is persistent anuria or severe oliguria, gross hematuria at the onset, and flank pain early in the course of the disease. These manifestations, however, are not specific and may be found in association with other forms of acute renal failure, particularly acute tubular necrosis. Bilateral cortical necrosis should be suspected, however, when acute anuria occurs in a multiparous woman during the last trimester of pregnancy, when accompanied by vaginal bleeding, toxemia, or pre-existing hypertension. The disorder should be considered in acute renal failure in children, especially when diarrhea, vomiting, and dehydration are part of the clinical picture. The diagnosis should also be entertained in cases of acute renal failure associated with septic shock.[13] Although the history and clinical course is suggestive of the diagnosis, the

roentgenographic findings may substantiate this impression. Gjørup and his coworkers[10], along with Margolin[14], feel that the appearance of a rim of renal cortical calcification, when associated with a compatible history and clinical course, is diagnostic of bilateral cortical necrosis.

ROENTGEN FINDINGS

Early in the course of bilateral renal cortical necrosis the kidneys may enlarge. This is followed by a variable degree of renal shrinkage. In our case the shrinkage occurred primarily during the first few months following the acute episode. However, further reduction in the size of the left renal outline was noted on roentgenograms taken nineteen months later. The faint cortical calcification, which has the appearance of penciling-in of the kidney margins, appears about one and one-half to two months after the onset of the disease. Both an anatomic and a pathologic basis exist for the appearance of this type of calcification. The normal renal cortex measures 5 to 8 mm. in thickness. Since a subcapsular zone as well as approximately one-third to one-half of the inner cortex are usually spared of necrosis,[7] it follows that only a relatively thin intervening layer of necrotic tissue is susceptible to calcification. Irregularity of the renal contour as well as interruption of the cortical calcification may occur. This is a reflection of the patchy distribution of the necrosis. The faint calcifications may be difficult to see on conventional roentgenograms of the abdomen, and laminagraphy may demonstrate the calcification to better advantage (Fig. 2, *A* and *B*). Laminagraphy is, therefore, recommended in suspected cases of renal cortical necrosis. In our case intravenous pyelography nineteen months after the acute episode revealed prompt excretion bilaterally with only moderate concentration of the contrast material (Fig. 3, *A* and *B*). The pelvicalyceal systems appeared normal.

Calcification somewhat similar to that observed in renal cortical necrosis has been reported in chronic glomerulonephritis, and on postmortem kidney roentgenograms of a case in which there were widespread osteolytic metastases to bone.[3] The history will serve to differentiate these cases from cortical necrosis. A number of conditions and substances have been associated with the production of renal calcification.[1,3,12] Examples of these are hyperparathyroidism, milk-alkali syndrome, renal tubular acidosis, sarcoidosis, chronic pyelonephritis, sulfonamide tubular damage and others.[9] To our knowledge the nephrocalcinosis associated with these conditions has not been confined, roentgenographically at least, to the renal cortex, and therefore they do not offer a problem in the differential diagnosis of bilateral cortical necrosis.

SUMMARY

A case of bilateral renal cortical necrosis is presented. Certain etiologic, clinical, and differential diagnostic features of this condition are discussed. The literature on bilateral renal cortical necrosis has been reviewed with special attention directed to the appearance of a characteristic type of renal calcification detectable roentgenographically. The present paper stresses the importance of this calcification in the separation of bilateral renal cortical necrosis from other causes of acute renal failure.

Stanley H. Nedelman, M.D.
Department of Radiology
Cincinnati General Hospital
Cincinnati 29, Ohio

We are indebted to Drs. Benjamin Felson, Edward A. Gall, and E. Gordon Margolin for their encouragement and invaluable assistance.

REFERENCES

1. Allen, A. C. The Kidney. Grune & Stratton, New York, 1951, pp. 220–224, 472–478.
2. Alwall, N., Erlanson, P., Tornberg, A., Moëll, H., and Fajers, C. Two cases of gross renal cortical necrosis in pregnancy with severe oliguria and anuria of 116 and 70 days respectively; clinical course, roentgenological studies of kidneys (size, outlines and califications), and post-mortem findings. *Acta med. scandinav.*, 1958, *161*, 93–98.

3. Arons, W. L., Christensen, W. R., and Sosman, M. C. Nephrocalcinosis visible by x-ray associated with chronic glomerulonephritis. *Ann. Int. Med.*, 1955, *42*, 260–262.
4. Boucot, N. G., Guild, W. R., and Merrill, J. P. Bilateral renocortical necrosis: with recovery; report of case. *New England J. Med.*, 1957, *257*, 416–418.
5. Case Records of the Massachusetts General Hospital, Case No. 44241. *New England J. Med.*, 1958, *258*, 1215–1223.
6. DeGraeff, J., and DeBaan, P. Bilateral renal cortical necrosis; recovery of urinary output after anuric period of 31 days. *Acta med. scandinav.*, 1959, *163*, 341–348.
7. Duff, G. L., and More, R. H. Bilateral cortical necrosis of kidneys. *Am. J. M. Sc.*, 1941, *201*, 428–450.
8. Dunn, J. S., and Montgomery, G. L. Acute necrotizing glomerulonephritis. *J. Path. & Bact.*, 1941, *52*, 1–16.
9. Geraci, J. E., Harris, H. W., and Keith, N. M. Bilateral diffuse nephrocalcinosis; report of 2 cases. *Proc. Staff Meet. Mayo Clin.*, 1950, *25*, 305–315.
10. Gjørup, S., Killmann, S. A., and Thaysen, J. H. Bilateral renal cortical necrosis; case followed during 51 days by means of hemodialytic treatment. *Acta med. scandinav.*, 1957, *158*, 47–54.
11. Gormsen, H., Iversen, P., and Rasschou, F. Kidney biopsy in acute anuria with case of acute bilateral cortical necrosis. *Am. J. Med.*, 1955, *19*, 209–221.
12. Hepler, O. E., and Simonds, J. P. Experimental nephropathies; calcification and phosphatase in kidneys of dogs poisoned with mercury bichloride, potassium dichromate and uranyl-nitrate. *Arch. Path.*, 1945, *40*, 37–43.
13. Lauler, D. P., and Schreiner, G. E. Bilateral renal cortical necrosis. *Am. J. Med.*, 1958, *24*, 519–529.
14. Margolin, E. G. Personal communication.
15. Moëll, H. Gross bilateral renal cortical necrosis during long periods of oliguria-anuria; roentgenologic observations in two cases. *Acta radiol.*, 1957, *48*, 355–360.
16. Mortensen, J. D., Baggenstoss, A. H., Power, M. H., and Pugh, D. G. Roentgenographic demonstration of histologically identifiable renal calcification. *Radiology*, 1954, *62*, 703–712.
17. Ober, W. E., Reid, D. E., Romney, S. L., and Merrill, J. P. Renal lesions and acute renal failure in pregnancy. *Am. J. Med.*, 1956, *21*, 781–810.
18. Sheehan, H. L., and Moore, H. C. Renal Cortical Necrosis and the Kidney of Concealed Haemorrhage. Charles C Thomas, Publisher, Springfield, Ill., 1953.
19. Wahle, G. H., Jr., and Muirhead, E. E. Bilateral renal cortical necrosis in a child associated with incompatible blood transfusion. *Texas J. Med.*, 1953, *49*, 770–775.
20. Wells, J. D., Margolin, E. G., and Gall, E. A. Renal cortical necrosis: Clinical and pathologic features in 21 cases. To be published.

URETHROGRAPHY IN THE ADULT MALE*

By JAMES W. WILLWERTH, M.D.†
READING, PENNSYLVANIA

FOR the past ten years, the subject of adult male urethrography has received little attention in the American radiologic literature. Several papers have considered the newer contrast media,[3-7,11,14] and specific applications for adult urethrography have been reported,[9,12,13,15] but only a few general reviews of this subject have been published.[10,14,16] It is the purpose of this paper to review these general considerations and our experience with retrograde urethrography, and perhaps to stimulate more general acceptance and usage of this simple and valuable diagnostic procedure.

It must be emphasized that retrograde urethrography as described in this presentation is of value only in the determination of disease processes which produce anatomic change in the male urethra, and that the procedure by itself is of limited value in the study of the physiology or pathophysiology of the lower urinary tract. These latter processes are best examined by voiding cystourethrography, preferably with a rapid cassette changer or cinefluorography. We feel that the infant male urethra is also best examined roentgenologically by using voiding cystourethrography, although there are instances when the retrograde study in conjunction with the voiding study is necessary to establish the diagnosis in a young patient.

CONTRAST MEDIA

Since Cunningham performed the first contrast visualization of the urethra with argyrol in 1910,[1] there has been a continuing search for a satisfactory medium for urethrography. The use of aqueous contrast media is attended by very few complications, but these media are not sufficiently viscous to distend the posterior urethra for adequate visualization of this structure. The oily media possess the desired viscosity but instances of pulmonary oil embolism have been reported.[17] Barium-water suspensions are also unsuitable for retrograde urethrography.[8] Thixokon (Mallinckrodt) is a water soluble medium with sufficient viscosity to distend all portions of the urethra and we believe it is the medium of choice. We first began to use thixokon in 1956 on an investigational basis. Since that time almost 200 urethrographic examinations have been performed without a single instance of toxic reaction or side effect, although upon several occasions the medium has inadvertently been extravasated into the corpus spongiosum and pelvic veins.

A comparison of thixokon urethrograms with those made previously with other media shows that the thixokon studies, almost without exception, demonstrate the entire urethra, whereas it was often impossible to force other media through the external sphincter. This may be due to the fact that thixokon is less irritable and allows greater pressures to be applied to the sphincter.

It has been our experience that thixokon possesses greater radiopacity than other aqueous media and therefore produces roentgenograms of superior technical quality. This greater opacity may occasionally result in missing a small radiolucent filling defect within the urethral lumen; this disadvantage may be overcome by the use of high kilovoltage techniques.

PROCEDURE

The equipment necessary to perform retrograde urethrography is quite simple, and, with the possible exception of the Brodny[2] instrument, is readily available in

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27-30, 1960.

† Present address: Department of Radiology, The Hahnemann Medical College and Hospital of Philadelphia, Philadelphia, Pennsylvania.

any radiologic department. The procedure itself is performed in a period of time rarely exceeding twenty minutes. A preliminary survey roentgenogram is made and examined for positioning and technique. The Brodny instrument is then attached and with constant pressure 20 to 25 cc. of medium is *slowly* injected. With experience, it is possible to "feel" when the hydrostatic pressure of the medium has overcome the resistance of the external sphincter and allowed the medium to fill the posterior urethra. At this time, four roentgenograms are made using the anteroposterior projection, each oblique projection with the patient in the "bicycle riding" position, and an evacuation film after removal of the conical tip portion of the Brodny instrument. The patient is asked to void as this last exposure is made.

For several years, our urethrograms were made with fluoroscopic guidance, employing various pressure maneuvers in an attempt to study the physiology as well as the anatomy of the lower urinary tract. This practice has now been discontinued because it was found to add little to the diagnostic usefulness of the retrograde study. In addition, fluoroscopy made the procedure technically more difficult and increased the exposure to both the patient and the operator. With the present four film technique and the use of the Brodny instrument, the exposure to the operator's hands is less than 15 mr per examination. We feel that this small exposure does not warrant the use of cumbersome lead gloves, although the operator does protect his body with a fluoroscopic lead apron.

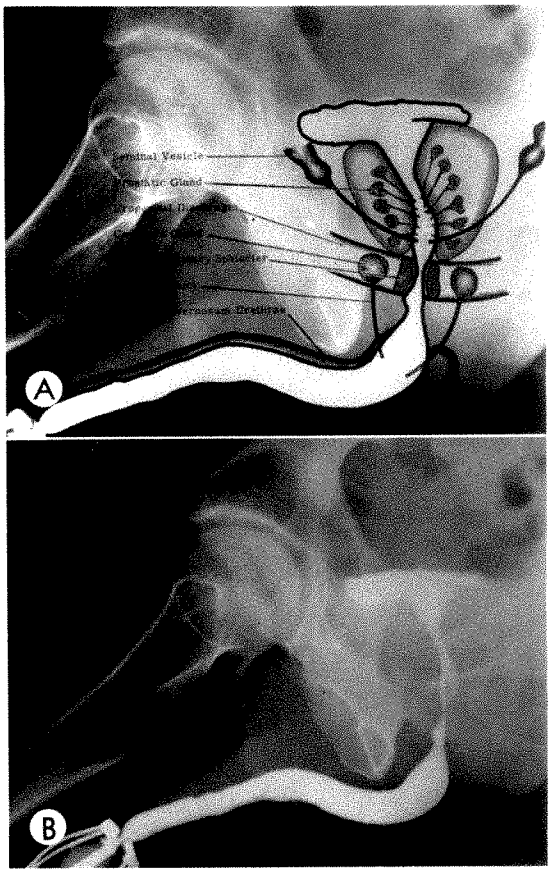


Fig. 1. (*A*) Superimposed diagram showing normal anatomy. (*B*) Normal urethrogram.

REVIEW OF NORMAL ANATOMY

The male urethra can be considered to be divided into three parts (Fig. 1*A*). The posterior urethra is that part which is surrounded by the lobes of the prostate gland and for that reason it is frequently called the prostatic urethra. This segment of the urethra averages 2.5 to 3.0 cm. in length and in cross section is crescent-shaped. The posterior elevation is produced by the verumontanum which, together with the urethral crests, extends the entire length of the posterior urethra. The seminal vesicles extend obliquely through the lateral lobes of the prostate and empty as the ejaculatory ducts on the apex of the verumontanum. On either side of the veru are the orifices of the ducts which drain the numerous prostatic glands.

The membranous urethra is that segment which lies within the layers of the urogenital diaphragm. Surrounding this part of the urethra is the external urinary sphincter. Cowper's glands are also located within the urogenital diaphragm, although the ducts from these glands enter the urethra in its bulbous portion.

The anterior urethra extends from the external urinary sphincter to the urethral meatus and is surrounded for almost its



FIG. 2. Uncomplicated anterior urethral stricture of venereal origin. The irregularity and lack of distensibility of the entire anterior urethra indicate chronic infection. A second, large-caliber stricture is present.

entire length by the corpus spongiosum. The anterior urethra is subdivided into the bulbous, or fixed, portion and the penile portion distal to the penoscrotal junction. Numerous small submucosal glands empty into the anterior urethra along its distal two-thirds.

Figure 1*B* illustrates the normal appearance of the urethra as seen on the urethrogram. The verumontanum produces a radiolucent filling defect in the posterior urethra. The membranous urethra is the narrowest part and is demarcated by the more distensible portions of the urethra just proximal and distal to it. The anterior urethra is normally smooth in contour and quite distensible. The area of narrowing just distal to the membranous urethra is called the musculus compressor nudae[10] and must not be mistaken for the membranous urethra itself or for an anterior urethral stricture. In the normal individual, none of the glandular structures or their ducts are visualized.

INDICATIONS

In general, the indication to us for retrograde urethrography in the adult male is the presence of any lower urinary tract symptoms which are not readily explained by the usual diagnostic modalities of history, physical examination, laboratory studies, and endoscopy. Some authors advocate urethrography for all adult males with lower urinary tract symptoms and we are in agreement with their opinion. Unfortunately, we have found that, in our hospital, routine urethrography is not feasible for both administrative and economic reasons and we have had to limit the study to those patients in whom some diagnostic difficulty exists.

The specific indications for urethrography will be considered separately with illustrative cases.

ILLUSTRATIVE CASES

CASE 1 (Fig. 2). This fifty-six year old male had been experiencing frequency, nocturia, dysuria, and dribbling for many years. These symptoms had been progressing in severity and just before urethrography was performed he had been admitted to the hospital in acute urinary retention. The admission diagnosis was acute retention secondary to prostatic hypertrophy. When attempts to catheterize the patient were unsuccessful, a urethrogram was obtained. Upon later questioning, the patient admitted social indiscretions as a youth.

Comment. Urethrography is indicated in all cases of urethral stricture because it not

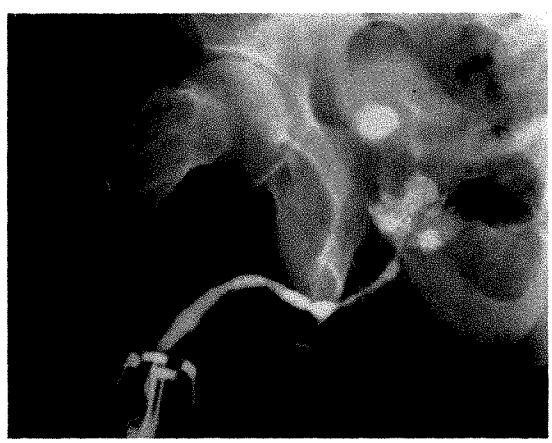


FIG. 3. Multiple anterior urethral strictures of venereal origin complicated by a urethrocutaneous fistula. Note the other stigmata of anterior stricture: dilated, infected Cowper's ducts and prostatic glands; false passages from attempted dilatations; contracted vesical neck caused by chronic infection.

only establishes the diagnosis of stricture but also demonstrates the location, number, and extent of the strictures, as well as the presence or absence of any of the complications of urethral stricture.

Case II (Fig. 3). This sixty-nine year old male had been treated for many years with periodic dilatations for urethral stricture. Shortly before this urethrogram was made, the patient developed a perineal abscess which promptly resulted in a urethrocutaneous fistula. Upon admission to the hospital, he was found to be unable to pass urine through the urethral meatus but was voiding without difficulty through the perineal fistula. The patient is now well following urethroplasty, suprapubic prostatectomy, and Y-plasty of the vesical neck.

Comment. Urethrography is necessary in all patients with urethral fistulae to determine the etiology of the fistula as well as to aid in planning the proper surgical approach. This study is also the only available method for examining the urethra proximal to the most distal urethral stricture present.

Case III (Fig. 4). This forty-eight year old male had had two transurethral resections prior to this urethrogram. These had been performed twelve and five years previously but had failed to relieve the symptoms of frequency, urgency, and nocturia. Proper resection of the previously unresected adenoma provided complete relief of symptoms.



Fig. 4. Residual prostatic tissue following incomplete transurethral resection. The unresected adenoma is exerting a ball-valve effect as it lies within the prostatic fossa.



Fig. 5. Sclerosis of the bladder neck following transurethral resection. Note the balloon-like dilatation of the prostatic fossa caused by the retrograde injection of opaque medium. The seminal vesicles are filled and one large false passage is present.

Case IV (Fig. 5). This sixty-nine year old male had had a transurethral resection for symptoms of prostatism five years prior to this study. He was symptomatically improved for several months following the resection. The frequency and nocturia then recurred and gradually became more severe than they had been before his operation. A Y-plasty of the contracted, sclerotic bladder neck was planned, but the patient refused further surgery and expired in uremia.

Comment. Cases III and IV demonstrate the value of urethrography in the post-prostatectomy patient who continues to have obstructive symptoms. We believe that all such patients should have urethrography performed before a second resection is attempted.

Case V (Fig. 6). This fifty-two year old male suddenly developed profuse, painless urethral bleeding one week before this study was done. His urinary function was not impaired and he denied any previous urinary symptoms. Endoscopy after spontaneous cessation of the bleeding revealed no abnormalities. The urethral diverticulum was excised through a perineal incision and the patient is again asymptomatic.

Comment. Urethrography is indicated in all patients with urethral bleeding and no



FIG. 6. True (congenital) urethral diverticulum. The urethral bleeding was apparently caused by rupture of the distal end of the diverticulum into the bulbous urethra. The proximal connection between the diverticulum and the urethra was not demonstrated by this study. At surgery, this connection was found just proximal to the membranous urethra.

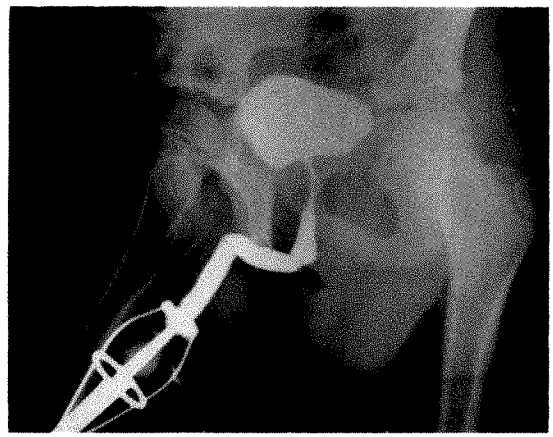


FIG. 7. A well-formed, large caliber, anterior urethral stricture six weeks following definitive repair of a severed urethra.

history of recent trauma, although it is well to wait until the bleeding has subsided before attempting the procedure. In this patient, neither of the orifices of the diverticulum was visualized by endoscopy and the diagnosis was in doubt until urethrography was performed.

CASE VI (Fig. 7). This five year old male child fell astride a bicycle injuring the perineum just dorsal to the scrotum. Immediately after the injury, there was gross urethral bleeding and he was unable to void. The urethra was surgically repaired over a splinting catheter passed through a cystotomy incision. This urethrogram was made six weeks following injury and operation at which time the patient was asymptomatic, although a well-formed stricture is already present.

Comment. Urethrography is a routine part of the postoperative management of urethral injuries resulting from external trauma. The study demonstrates the presence or absence of stricture formation at that time and serves as a baseline for further studies should the patient develop obstructive symptoms in the future.

CONTRAINDICATIONS

Contraindications to retrograde urethrography are few. It is our opinion that acute urethral injury from external trauma is one of these contraindications because the diagnosis is evident by history and physical examination alone and because the extravasated opaque medium may in itself contribute to stricture formation. In the unusual case of this kind in which urethrography may be necessary, an aqueous contrast medium should be used.

It has been our experience that most urethrograms made shortly after instrumentation of the urethra were not of diagnostic quality due to extravasation of the contrast medium through small mucosal tears resulting from the previous internal trauma. The study is therefore best performed before attempting catheterization or endoscopy.

Urethrography should be postponed in the presence of acute infection because of the possibility of producing vascular dissemination of the infecting organisms.

SUMMARY

We believe that urethrography is a much neglected but quite useful diagnostic procedure. It is hoped that this review will stimulate increased interest in and applica-

tion of this method for investigation of the lower urinary tract.

Department of Radiology
The Hahnemann Medical College and
Hospital of Philadelphia
Philadelphia, Pennsylvania

REFERENCES

1. BEARD, D. E., GOODYEAR, W. E., and WEENS, H. S. Radiologic Diagnosis of the Lower Urinary Tract. Charles C Thomas, Publisher, Springfield, Ill., 1952
2. BRODNY, M. L. New instrument for urethrography in male. *J. Urol.*, 1941, *46*, 350–354.
3. DENMAN, J. Thixokon: improved urethrographic medium. *J. Urol.*, 1957, *78*, 93–96.
4. GLENN, J. F. Thixokon cystourethrography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 631–634.
5. GODDARD, D. W. Experiences with thixokon: new urethrographic medium. *J. Urol.*, 1959, *81*, 225–226.
6. KAUFMAN, J. J., and RUSSELL, M. Cystourethrography: clinical experiences with newer contrast media. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *75*, 884–892.
7. KAUFMAN, J. J. Experiences with thixokon: aqueous, thixotropic urethrographic medium. *J. Urol.*, 1957, *78*, 188–191.
8. KJELLBERG, S. R., ERICSSON, N. O., and RUDHE, U. The Lower Urinary Tract in Childhood. The Year Book Publishers, Inc., Chicago, 1957.
9. MICKALOWSKI, E., and KRAKOWSKI, J. Diverticula of anterior urethra in man. *Urol. Internat.*, 1957, *5*, 301–309.
10. MORALES, O., and ROMANUS, R. Urethrography in male. *J. Urol.*, 1955, *73*, 162–171.
11. MORALES, O., and ROMANUS, R. Urethrography in male with highly viscous, water-soluble contrast medium, umbradil-viscous U. *Acta radiol.*, 1952, Suppl. 95.
12. ORAVISTO, K. J., and SCHAUMAN, S. Urethrocystography in differential diagnosis of prostatic cancer. *J. Urol.*, 1956, *75*, 995–999.
13. PATE, V. A., JR., and BUNTS, R. C. Urethral diverticula in paraplegics. *J. Urol.*, 1951, *65*, 108–125.
14. THOMPSON, I. M. Safe contrast medium for urethrography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 627–630.
15. THUMANN, R. C., JR. Estimation of weight of hyperplastic prostate from cystourethrogram. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 593–595.
16. THUMANN, R. C., JR., and RANDALL, D. Cystourethrography in diagnosis of diseases of prostatic urethra. AM. J. ROENTGENOL. & RAD. THERAPY, 1950, *64*, 640–648.
17. ULM, A. H., and WAGSHUL, E. C. Pulmonary embolization following urethrography with oily medium. *New England J. Med.*, 1960, *263*, 137–139.

LIPOMA OF THE CORPUS CALLOSUM*

By GERTRAUD WOLLSCHLAEGER, M.D.,† PAUL B. WOLLSCHLAEGER, M.D.,† DON D. BRANNAN, M.D.,‡ *and* A. J. SEGAL, M.D.§
CLEVELAND, OHIO

LIPOMA of the corpus callosum is a rare intracranial midline lesion. The first pathologic description of this entity was presented by Rokitansky.[26] Other reports have since appeared in the literature.[2,3,9,12,19,25,27,32,34] We have been able to find only 13 out of about 50 previously reported cases that had been correctly diagnosed prior to operation or death. The first roentgen diagnosis on plain skull roentgenograms was made by Sosman[31] in 1939.

The purpose of this paper is to present an additional case of lipoma of the corpus callosum which was diagnosed on plain skull roentgenograms prior to death and to show the distribution of calcification in the lesion with the aid of roentgenograms of brain slices.

PATHOGENESIS

The most commonly accepted hypothesis of the pathogenesis of lipoma of the corpus callosum is that of Krainer,[20] and of Wassermann.[36] They believe that lipoma develops from persistent primitive meningeal tissue, and state that this occurs in the leptomeningeal cisterns. This hypothesis was concurred in by Merkel,[22] List *et al.*,[21] and Vonderahe and Niemer.[35] These authors assume that the development of lipoma occurs in very early embryonic life (about the second fetal month) and persists into maturity.

The interference with the development of the corpus callosum and the adjacent structures by the persistence of this primitive tissue has been discussed by de Morsier.[23] It seems very likely that agenesis, or partial agenesis, of the corpus callosum is the consequence of this congenital lesion. In our opinion the interference with development is dependent upon the location of the persistent primitive tissue. Our belief is based on the fact that the splenium and septum pellucidum are absent in the presence of a lipoma, as seen in our case. Similar findings were reported by Würth,[38] List *et al.*,[21] and Huddleson,[15] who observed total agenesis of the corpus callosum, and Juba,[17] Huebschmann,[16] Rubinstein,[27] von Sury,[32] Benjamin,[2] Ehni and Adson,[11] Schnürer and Zettergren,[28] Kirkbride,[19] Dyke,[10] Ernst,[12] and Sheinmel and Lawrence,[29] who were able to demonstrate partial agenesis of the corpus callosum in the presence of lipoma. Sosman[31] also reported agenesis of the corpus callosum in his 3 cases.

A few cases are described by Wolfson *et al.*[37] without associated congenital anomalies. In these cases the lipoma was situated above the corpus callosum, tapering in anteroposterior direction. These various findings support the belief that the original localization of the primitive tissue is mainly responsible for associated anomalies, and that one is dealing with a secondary agenesis of the corpus callosum. The latter must be clearly distinguished from primary agenesis, as shown in 2 case reports by Sheinmel and Lawrence[29] and Zellweger.[39] Baker and Graves[1] did not find any lipoma in their 82 cases of partial agenesis of the corpus callosum, nor did Bunts and Chaffee[7] in their 15 cases.

Lipoma of the corpus callosum is a benign tumor composed of mature fat and fibrous tissue and resembles the typical lipoma of any other region of the body. It is not infiltrative and does not possess atypical cells or abnormal mitoses.

Lipoma occurs with equal frequency in the male and female. The age distribution

* From the Department of Radiology and the Division of Pathology, Saint Luke's Hospital, Cleveland, Ohio.
† Residents in Radiology.
‡ Head of the Department of Radiology.
§ Director of the Division of Pathology.

varies from thirty months[24] to seventy-five years.[13]

Lipoma of the corpus callosum may be asymptomatic. Signs and symptoms, when present, are those of a midline lesion of the brain and are not specific for lipoma. Persistent severe headaches, grand mal seizures, hemiparesis and mental changes have been variously reported.[5]

A definitive diagnosis of lipoma of the corpus callosum can be made, during life, from the characteristic roentgenographic appearance.[21]

REPORT OF A CASE

A seventy-one year old white male (Unit No. C-58420) was admitted on October 26, 1959 with a chief complaint of persistent headaches since 1944, associated with occasional dizzy spells and weakness of legs. The positive physical findings were limited to the central nervous system and indicated a spinal cord lesion. Myelography revealed a block at the level of the eighth and ninth dorsal segments. The block was considered to be secondary to a metastatic tumor. Complete clinical data of this patient will appear in another report.[33]

Roentgenograms of the skull were taken on admission. In the posteroanterior view there were symmetric parasagittal curvilinear densities with lateral convexities. The distance between these densities measured about 2 cm. (Fig. 1*A*). A curvilinear density with superior convexity was observed in the lateral view in the region of the corpus callosum. Inferior to this was a zone of relative radiolucency (Fig. 1*B*). A diagnosis of lipoma of the corpus callosum was made (P.B.W.). A repeat roentgenographic examination of the skull was performed about two weeks later and revealed the same findings.

Pneumoencephalographic and arteriographic studies were not performed because of the patient's general condition.

The patient was treated symptomatically and was discharged to a nursing home on November 27, 1959 and died there on December 16, 1959.

Necropsy revealed that the patient had a bronchogenic carcinoma with metastasis to the spinal dura, producing the block and cord compression that resulted in a cord bladder, complicated by suppurative pyelonephritis. For the purposes of the present report we will limit our remarks to the description and discussion of the brain.

Roentgenograms of the formalin-fixed brain were made prior to dissection so as to visualize the ventricular system without the benefit of air injection. There were dilatation of the posterior horns of the lateral ventricles, more so on the left, and marked separation of the frontal horns. Calcification within the tumor was seen in various projections.

The brain was dissected after ten days' fixa-

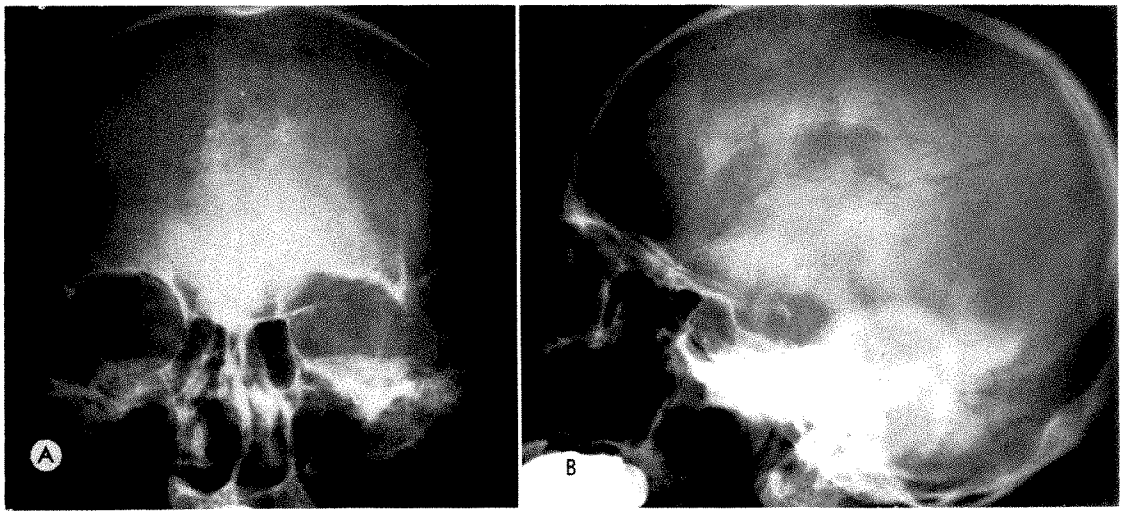


Fig. 1. (*A*) Posteroanterior roentgenogram of skull. Symmetric curvilinear densities with lateral convexities are present in a parasagittal position. (*B*) Lateral roentgenogram of skull demonstrates the curvilinear density with the superior convexity and the relatively radiolucent area, inferior to the dense line.

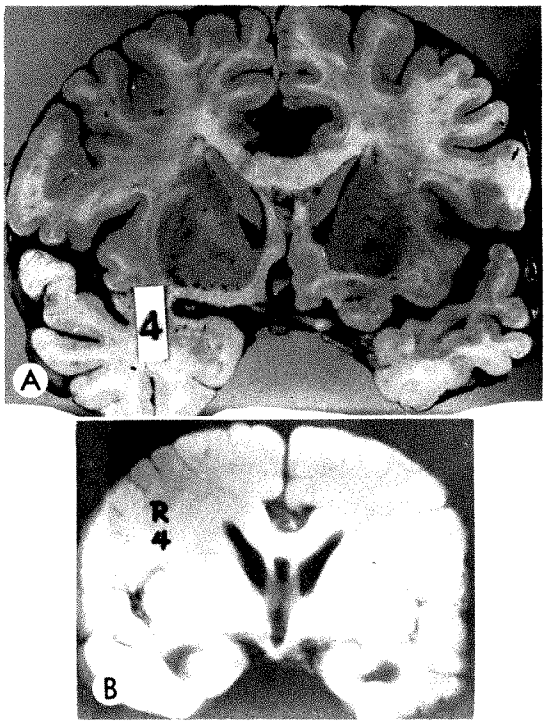


FIG. 2. (*A*) Coronal section and (*B*) roentgenogram through midportion of anterior horns of the lateral ventricles. There is definite dilatation of the sulcus corporis callosi. The septum pellucidum is absent and the fornices columnae are attached to the corpus callosum. At this level, the lipoma is only a thin layer over the corpus callosum. The calcified bands accompany the tumor laterally into the adjoining brain substance.

tion in 10 per cent formalin. It weighed 1,600 gm. The gyri were flattened and the sulci were slightly widened. The cerebral hemispheres were equal and symmetric. The cranial nerve distribution was normal. The arteries were in normal position. They were focally narrowed by atheromatous plaques. The pia mater was thickened and opacified.

There was an irregular, lobulated, yellow, fatty tumor involving the corpus callosum.The main body of the tumor within the corpus callosum measured 2.7×3.1×2.5 cm. There were several offshoots extending in various directions. One of these was a sheet, 1.9 cm. wide and 0.2 cm. thick, covering the surface of the corpus callosum from the genu to a point approximately 7 cm. posterior to the genu. This tissue covered the raphe, the striae longitudinales mediales and laterales and a small portion of the gyri cinguli. Another projection presented in the right lateral ventricle as a bulbous mass 1.2 cm. in diameter connected to the main mass by a stout pedicle occupying a dilated foramen of Monro. Still another projection of tumor presented as a cap over the third ventricle. The anterior cerebral arteries were thickened and were contained within the tumor tissue. The tumor appeared to be well defined, with a fine membranous capsule and was soft except for numerous discrete and confluent hard white calcified plaques. Some of the latter presented as tiny flecks in the capsule, while more concentrated masses were found in the superior portion of the corpus callosum and in the striae longitudinales laterales. There were anomalies of the corpus callosum, including incomplete development of the splenium, absence of the septum pellucidum and widening of the sulcus or cisterna corporis callosi. The cut surfaces of the corpus callosum presented a watered silk appearance caused by interlacing bands of pale yellowish-white fibrous tissue.

Photographs and roentgenograms of 8 coro-

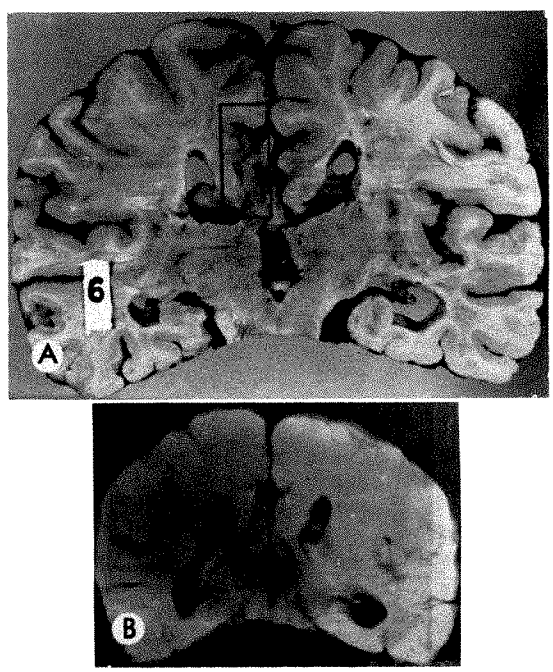


FIG. 3. (*A*) Coronal section and (*B*) roentgenogram through posterior third of body of lateral ventricle. The continuity of the corpus callosum is interrupted by the main mass of the lipoma. A projection of the tumor forms a cap over the dilated third ventricle while another projection extends into the right lateral ventricle. The fibrous tissue streaks in the corpus callosum are readily seen. *B* shows that the visible calcium still appears as a thin band adjacent to the sulcus corporis callosi. The rectangle outline in *A* indicates the location from which the section shown in Figure 4*A* was taken.

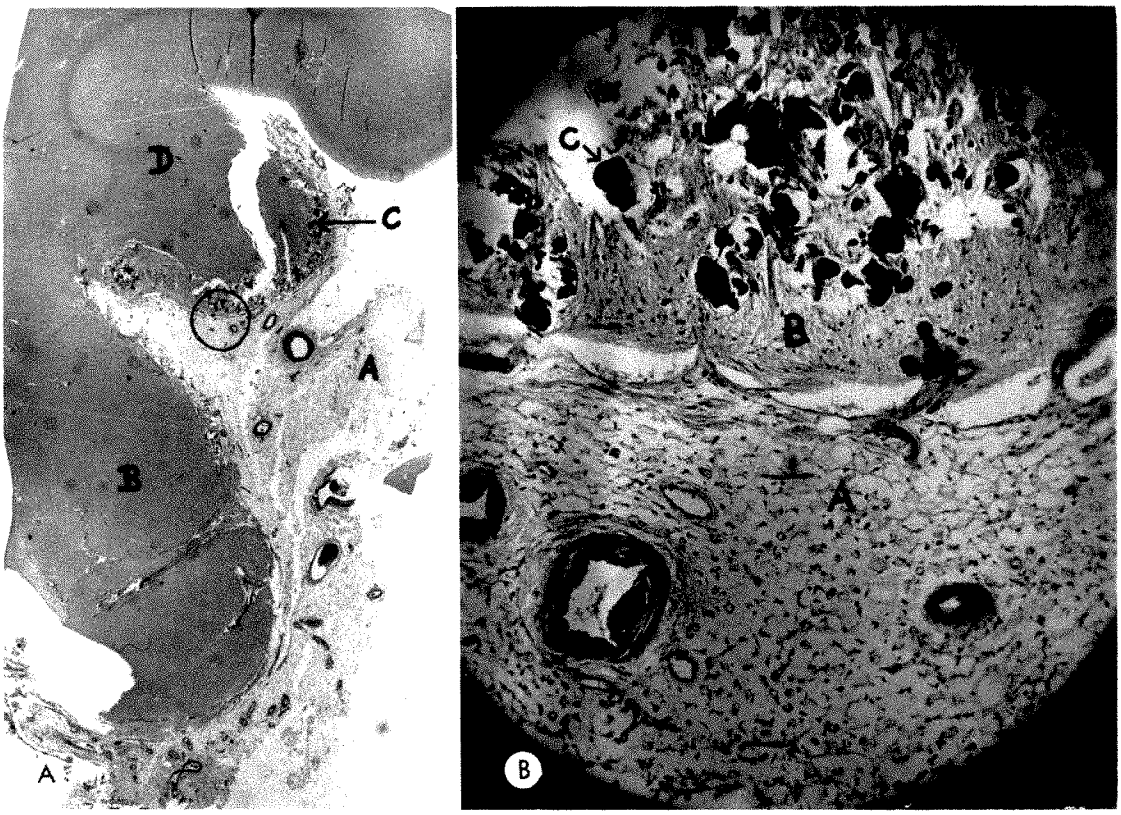


Fig. 4. (*A*) Low power view (5×) of rectangular section of Figure 3*A* shows adjacent corpus callosum and gyrus cinguli. A, lipoma; B, fragment of corpus callosum; C, calcification; D, gyrus cinguli. The area bounded by the circle is shown in *B*. (*B*) This demonstrates the mature adipose tissue of the lipoma with numerous thick-walled vessels. Note the deposition of calcium in the adjacent brain tissue. It is this calcification which is seen on roentgenograms (80×). A, fat tissue; B, brain tissues; C, calcification.

nal brain sections containing the tumor were taken. Only numbers 4 and 6 are included in this paper (Fig. 2, *A* and *B;* and Fig. 3, *A* and *B*). The others exhibited similar features.

Microscopic study showed the tumor composed of mature fat cells with abundant vascular support in the periphery and with fine collagenous stromal support producing incomplete lobulation. The continuity of the corpus callosum was interrupted, as previously described. The intermediate space was occupied by this same fatty tissue, in some fields bounded by coarse bands of dense collagenous tissue. The latter occurred also in irregular bands on the surface of the corpus callosum and within the corpus callosum, mostly around blood vessels. It is this tissue which was responsible for the coarsely trabeculated irregularities in the cut surface of the corpus callosum. Small and large calcified bodies were found mainly in the periphery of the fatty tumor and in the adjacent brain tissue (Fig. 4, *A* and *B*).

DISCUSSION

The roentgenographic features of lipoma of the corpus callosum are very characteristic but there is no uniformity in the reported location of the calcifications seen on the plain roentgenograms.

It has been variously stated that the calcifications are present in the tumor, in the capsule of the tumor, in the adjacent brain tissue, or in all of these locations simultaneously. This statement is certainly true from a histopathologic standpoint. However, the characteristic calcifications which are identifiable on the plain skull roentgenograms are chiefly located in the brain tissue bordering the sulcus corporis callosi, as seen in our case. The size and the concentration of the visible calcification are not related to the size of the tumor. The latter is variably described from pea-size to egg-size or as a

sheet spread over the surface of the corpus callosum. The pressure of the tumor on the highly sensitive adjoining brain substance affects the circulation. The amount and the intensity of calcium deposit are dependent upon the degree of circulatory disturbance. This conclusion has been arrived at by Bull.[6]

Roentgenograms of the individual coronal brain sections show calcific densities which produce the parenthesis-like bilateral densities visualized in the posteroanterior view of the skull. The continuity of the calcification in the anteroposterior direction along the superficial brain substance of the gyri cinguli is demonstrated on the lateral roentgenograms of the total brain.

We assume that the visible calcifications are, in most instances, in the adjoining brain tissue because of the uniformity of the characteristic roentgenographic features of published roentgenograms of cases of lipoma of the corpus callosum. The distance between the parenthesis-like calcification in the posteroanterior view depends upon the lesser or greater widening of the sulcus corporis callosi. Another observation favoring our hypothesis is the fact that the curvilinear density in the lateral view constantly appears with a convexity directed superiorly, and it has never been demonstrated with the convexity directed inferiorly.

The second pathognomonic sign, the so-called radiolucent area inferior to the curvilinear density in the lateral view, depends upon the size of the compact fatty tumor mass. This area may be a true radiolucency or a lesser degree of density in comparison to the surrounding brain.

Diagrams of plain roentgenograms of the skull of reported cases of lipoma of the corpus callosum are presented by Mullen and Hannan,[24] who noted the constancy of these calcified lines and of the radiolucency in some cases. The same is true in our case.

We would emphasize that no other intracranial midline lesion presents a similarly demarcated radiolucent zone and such typical curvilinear calcification. It is possible, thereby, to distinguish lipoma from primary agenesis of the corpus callosum, congenital cyst of the cavum septi pellucidi and tumors of the corpus callosum such as astrocytoma, oligodendroglioma and ependymoma, named in the order of their frequency.

Successful removal of the total lesion has not been reported.[4,8,14,18,21,24,36] Occasional attempts have been made to excise the benign tumor but had to be terminated because of bleeding due to the vascularity of the lipoma and the envelopment of the anterior cerebral arteries and their branches by the tumor.

The patient's condition and symptoms should and can be very well controlled by sedatives and anticonvulsants.[30]

SUMMARY

1. A case of lipoma of the corpus callosum has been presented.

2. The diagnosis was established prior to autopsy by plain roentgenograms of the skull.

3. Evidence has been presented to show that this is a congenital malformation.

4. This is the first case reported in which roentgenograms of the coronal brain sections were taken to localize the calcium distribution.

5. A brief review of the history, pathogenesis, pathology and differential diagnosis is presented.

Gertraud Wollschlaeger, M.D.
2838 E. 126th Street
Cleveland 20, Ohio

We wish to express our thanks to Mr. F. Beal and Mr. E. Jones for the photographic reproductions, and to Mrs. Lois A. Hagar, M.T. (A.S.C.P.) for the histologic preparations. We are also very thankful to Miss Betty Takiguchi.

REFERENCES

1. BAKER, R. C., and GRAVES, G. O. Partial agenesis of corpus callosum. *Arch. Neurol. & Psychiat.*, 1933, *29*, 1054–1065.

2. BENJAMIN, L. Beschreibung einer Knochen-

geschwulst im Gehirn. *Virchows Arch. path. Anat.*, 1858, *14*, 552–554.
3. BOSTROEM, E. Über die pialen Epidermoide, Dermoide und Lipome und duralen Dermoide. *Zentralbl. allg. Path. u. Path. Anat.*, 1897, *8*, 1–98.
4. BRANDENBURG, W., and KETZ, E. Lipom des Balkens. Ein lipoplastisches Meningeom. *Ärztl. Wchnschr.*, 1955, *10*, 366–369.
5. BREMER, F., BRIHAYE, J., and ANDRÉ-BALISAUX, G. Physiologie et pathologie du corps calleux. *Schweiz. Arch. Neurol. et Psychiat.*, 1956, *78*, 31–87.
6. BULL, J. cited by KETZ, E.[18]
7. BUNTS, A. T., and CHAFFEE, J. S. Agenesis of corpus callosum with possible porencephaly; review of literature and report of case. *Arch. Neurol. & Psychiat.*, 1944, *51*, 35–53.
8. CASCINO, J. P., CAKE, F., JACKSON, D., and KAPLAN, A. Lipoma of corpus callosum. *J. Internat. Coll. Surgeons*, 1958, *29*, 171–174.
9. COATS, J. A. Peculiar fatty growth of upper surface of corpus callosum. *Brit. M. J.*, 1874, *2*, 75.
10. DYKE, C. G. Roentgen ray diagnosis of skull and intracranial contents. In: Diagnostic Roentgenology. Thomas Nelson & Sons, New York, 1948, p. 33.
11. EHNI, G. J., and ADSON, A. W. Lipoma of brain; report of cases. *Arch. Neurol. & Psychiat.*, 1945, *53*, 294–304.
12. ERNST, P. Ein bügelformiges gemischtes Lipom auf dem Balken. Festschr. für J. Arnold, *Beitr. z. pathol. Anat. Suppl.*, 1905, *7*, 1–28.
13. FATTOVICH, G. Contributo allo studio dei lipomi del corpo calloso. *Riv. di pat. nerv.*, 1938, *52*, 310–320.
14. HUBER, K., HAMMER, B., and SEITELBERGER, F. Ein operiertes intrakranielles Lipom im Dach des 3. Ventrikels. *Wien. Ztschr. Nervenh.*, 1953, *7*, 104–114.
15. HUDDLESON, J. H. Ein Fall von Balkenmangel mit Lipomentwicklung in Defekt. *Ztschr. f. d. ges. Neurol. u. Psychiat.*, 1928, *113*, 177–192.
16. HUEBSCHMANN. Über einige seltene Hirntumoren. *Deutsche Ztschr. f. Nervenh.*, 1921, *72*, 205–224.
17. JUBA, A. Über einen mit Lipomatose verbundenen Fall von partiellem Balkenmangel. *Arch. f. Psychiat.*, 1937, *106*, 324–332.
18. KETZ, E. Zur Klinik und Therapie der sog. Balkenlipome. *Zentralbl. Neurochir.*, 1956, *16*, 80–84.
19. KIRKBRIDE, T. Lipoma of brain. *Path. Anat. Arb. Joh. Orth. z. Prof. Jubil.*, 1903, 515–519.
20. KRAINER, L. Die Hirn- und Rückenmarkslipome. *Virchows Arch. path. Anat.*, 1935, *295*, 107–142.
21. LIST, C. F., HOLT, J. F., and EVERETT, M. Lipoma of corpus callosum; Clinicopathologic study. AM. J. ROENTGENOL. & RAD. THERAPY, 1946, *55*, 125–134.
22. MERKEL, H. Zur Frage der Balkenlipome. Zugleich ein Beitrag zur Entwicklungsgeschichte des Balkens. *Ztschr. f. d. ges. Neurol. u. Psychiat.*, 1941, *171*, 269–277.
23. DE MORSIER, G. Études sur les dysraphies cranio-encephaliques. *Schweiz. Arch. Neurol. u. Psychiat.*, 1954, *74*, 309–361.
24. MULLEN, W. H., JR., and HANNAN, J. R. Roentgen diagnosis of lipoma of corpus callosum. *Radiology*, 1950, *55*, 508–516.
25. PARROT, J. Sur un cas de lipome de pie-mère cérébral. *Arch. de physiol. norm. et path.*, 1869, *12*, 443–447.
26. ROKITANSKY, C. Lehrbuch der Pathologischen Anatomie. Braunmüller, Wien, 1856, *2*, 468.
27. RUBINSTEIN, B. G. Über einen Fall von unvollständig fehlendem und durch Fettgewebe ersetztem Balken. *Frankfurt. Ztschr. f. Path.*, 1932–1933, *44*, 379–386.
28. SCHNÜRER, A. B., and ZETTERGREN, L. Lipom I. Corpus Callosum. *Nordisk Medicin Bd.*, 1959, *61*, 602–605.
29. SHEINMEL, A., and LAWRENCE, L. R. Agenesis of corpus callosum; lipoma of corpus callosum. *Radiology*, 1951, *57*, 15–24.
30. SMITH, C. D., and WEAVER, E. N. Lipoma of corpus callosum. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1953, *69*, 605–607.
31. SOSMAN, M. C. Discussion (p. 148) of paper by Echternacht, A. P., and Campbell, J. A. Midline anomalies of brain: their diagnosis by encephalography. *Radiology*, 1946, *46*, 119–131.
32. VON SURY, K. Ein gemischtes Lipom auf der Oberfläche des hypoplastischen Balkens. *Frankfurt. Ztschr. f. Path.*, 1907, *1*, 484–491.
33. TICICH, S. To be published.
34. VIRCHOW, R., Die krankhaften Geschwülste. 1863, *1*, 386–387.
35. VONDERAHE, A. R., and NIEMER, W. T. Intracranial lipoma; report of 4 cases. *J. Neuropath. & Exper. Neurol.*, 1944, *3*, 344–354.
36. WASSERMANN, F. Die Fettorgane des Menschen. Entwicklung, Bau und systematische Stellung des sogenannten Fettgewebes. *Ztschr. f. Zellforsch. u. mikr. Anat.*, 1925–1926, *3*, 235–328.
37. WOLFSON, J. J., SMITH, G. W., HANBERY, J. C., and LUTRELL, C. N. Lipoma of corpus callosum; report of 4 cases, 2 with cerebral anteriography. *Stanford M. Bull.*, 1957, *15*, 274–282.
38. WÜRTH, A. Ein Beitrag zur Histologie und Symptomatologie der Balkentumoren. *Arch. f. Psychiat. u. Nervenh.*, 1902, *36*, 651–657.
39. ZELLWEGER. Agénésis et lipomes du corps calleux. *Schweiz. Arch. Neurol. u. Psyschiat.*, 1956, *77*, 367–380.

ROENTGENOLOGIC FEATURES OF NEUROFIBROMAS INVOLVING THE GASSERIAN GANGLION*

By COLIN B. HOLMAN, M.D., *Section of Roentgenology*; IGNACIO OLIVE, M.D., *Fellow in Neurosurgery, Mayo Foundation; and* HENDRIK J. SVIEN, M.D., *Section of Neurologic Surgery*

MAYO CLINIC AND MAYO FOUNDATION†
ROCHESTER, MINNESOTA

NEUROFIBROMAS arising from the intracranial portion of the fifth nerve are rare. Approximately 43 cases had been reported in the literature prior to this series. The present report is a consideration of the roentgenologic features in the 13 cases reported by Olive and Svien[3] in 1957, plus another case encountered since; an additional 12 cases were reported by Schisano and Olivecrona[4] in 1960. Because such a study is necessarily retrospective in character, it should not be implied that the manifestations described are pathognomonic of this lesion. However, some interesting features occur that, when considered with the clinical evaluation of the patient, should strongly suggest the presence of a neurofibroma in this region.

These tumors of the trigeminal nerve exist primarily within the middle or the posterior cranial fossa or both fossae. Histologically, they resemble neurilemmomas of the acoustic nerve. Jefferson[1] pointed out that most tumors involving the gasserian ganglion actually arise from the pharynx and invade through the base of the skull, as judged by the clinical history or the presence of metastasis to the cervical lymph nodes or both. All such lesions have been excluded from this series, and pathologic confirmation of each tumor has been established.

The importance of the correct interpretation of the roentgenologic features arises from the paucity of initial clinical signs referable to involvement of the fifth nerve in many cases. Thus, in some instances, an earlier diagnosis of trigeminal neurofibroma may be suggested by roentgenologic means.

FINDINGS IN PRESENT STUDY

In this study, the roentgenograms and clinical records of 14 patients seen at the Mayo Clinic who had solitary neurofibromas of the trigeminal nerve were reviewed. None of these patients had any evidence of Recklinghausen's neurofibromatosis. The tumors were located primarily in the middle cranial fossa in 7 patients, in the middle and posterior fossae in 3 and in the posterior fossa in 4. Roentgenographic changes were not present in one patient in each category.

In one other patient, a tumor 2.5 cm. in diameter was an incidental finding at necropsy; roentgenograms of the head had not been made, but there was no evidence of bony erosion.

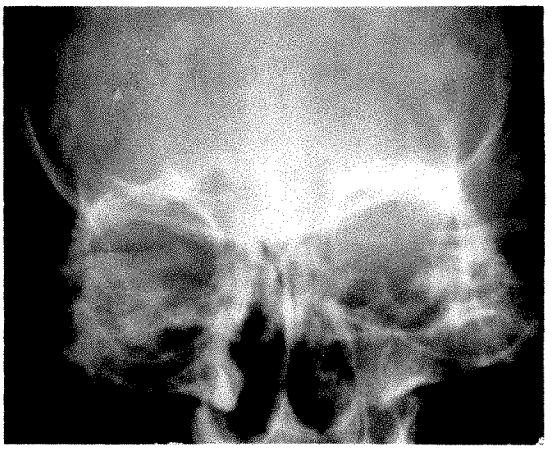


FIG. 1. Decalcification of the greater wing of the sphenoid on the right, with severe medial destruction of petrous portion of right temporal bone.

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

The bony changes accompanying the lesions in the middle fossa were those which might be expected to be associated with parasellar masses that expand slowly, and these changes corresponded to that portion of the middle fossa in which the tumor attained its maximal size. In 2 instances, the forward extension of the tumor resulted in decalcification of the greater wing of the sphenoid adjacent to the superior orbital fissure (Fig. 1). In another case, an extremely large tumor extended throughout the floor of the middle fossa medially and, therefore, eroded the side of the sella turcica and the anterior clinoid process as well as the base of the skull in the region of the foramen ovale; erosion of the optic canal and the medial portion of the petrous tip also was present (Fig. 2, *a* and *b*). Two other large tumors were associated with erosion of the side of the sella turcica, including the posterior clinoid processes. Two patients gave evidence of a soft-tissue tumor in the region of the pterygoid fossa and posterior nasopharynx (Fig. 3).

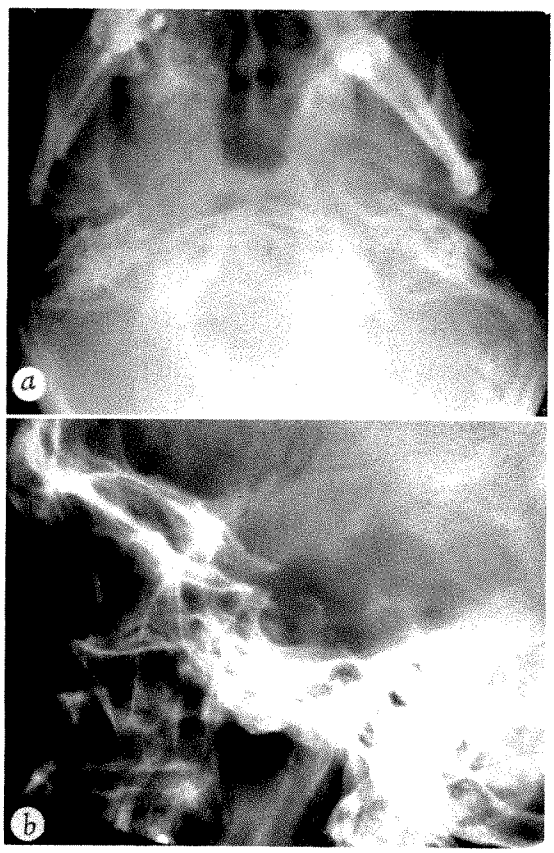


FIG. 2. (*a*) Extensive destruction of floor of right middle fossa, including right petrous tip. (*b*) Lateral view in same case, showing erosion of inferior aspect of right anterior clinoid and region of optic canal.

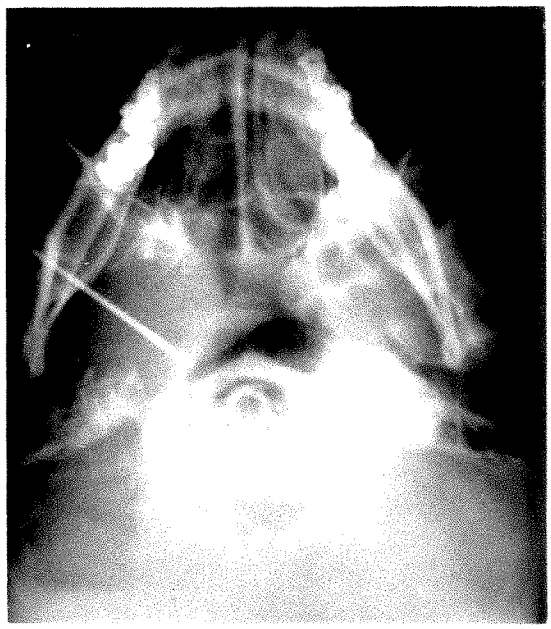


FIG. 3. Large soft-tissue mass extending into nasopharynx on right. The bony floor of the right middle fossa is destroyed. The shadow of a hairpin is superimposed over this region.

The most common and most significant osseous change involved the tip of the petrous bone. Erosion of the medial aspect of the petrous tip, leaving a rather sharp medial border, was noted in all 8 cases in which the petrous bone was involved (Fig. 4*a*). In each case, the destruction was medial to and did not involve the internal acoustic meatus. Although it has been reported that such destruction usually involves the superior portion of the tip, this was conspicuously lacking in at least one of our cases (Fig. 4*b*).

The floor of the middle fossa was known to be destroyed in 2 of the 3 cases in which the tumor was located partly in the middle fossa and partly in the posterior fossa. Erosion of the tip of the petrous bone medial to the internal acoustic meatus was

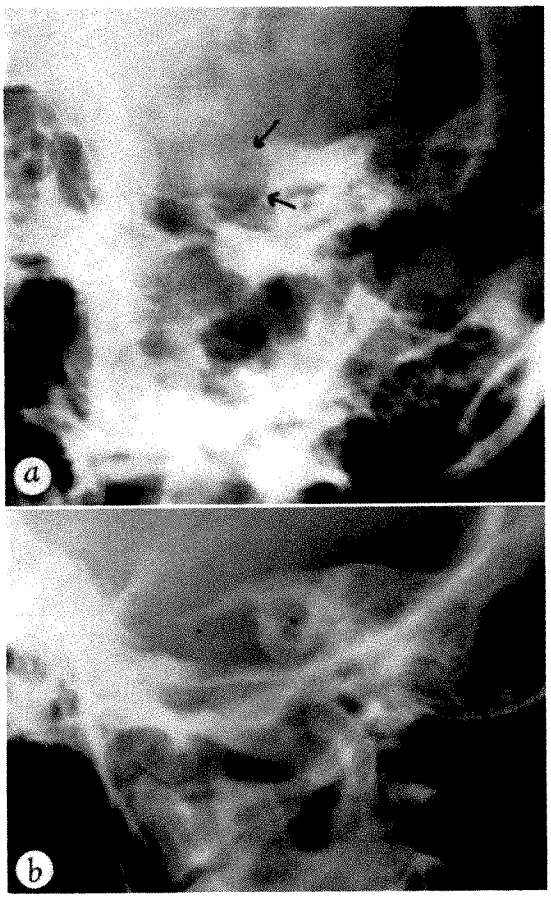


FIG. 4. (*a*) Destruction of left petrous tip medial to internal acoustic meatus, with well-defined, sharp, medial border (arrows). (*b*) Destruction of left petrous tip, with sparing of superior aspect.

present in both of these instances. These tumors may show the characteristics of growth in either of these cranial fossae. In those instances in which the bulk of the tumor was in the middle fossa, the findings previously noted prevailed.

When the mass was almost entirely within the posterior fossa, the roentgenologic characteristics were similar to those of a tumor at the cerebellopontine angle. However, as already noted, erosion of the petrous tip uniformly occurred medial to the acoustic meatus, which is not the usual feature accompanying neurofibromas of the eighth nerve. The rather sharp outline of the petrous tip at the site of the erosion helps to distinguish this lesion from the usual type of meningioma in the cerebellopontine angle. The appearance is not unlike that sometimes associated with erosion from an epidermoid tumor (Fig. 5).

Special Contrast Examination. Cerebral angiography was undertaken in 2 cases because the changes involving the sella turcica and parasellar region suggested the presence of a parasellar aneurysm of the internal carotid artery. In one of these studies, the internal carotid artery appeared to be compressed as it emerged from the region of the carotid foramen, suggesting compression by a mass immediately adjacent to it (Fig. 6). Both patients had some elevation of the vessels of the sylvian complex on the affected side, which indicated a mass in the middle fossa. It was our original impression on review of this material that the basilar vein of Rosenthal might have been elevated significantly. However, after a comparison was made with a number of angiograms in which lesions in this region were not present, it is our opinion that the basilar vein has a somewhat variable location and that it appeared to be in about the same position in many other cases that it was in these 2 instances.

Ventriculography was undertaken in one patient, who had a mass in both the middle and posterior fossae. Elevation of the temporal horn with amputation of its tip was demonstrated (Fig. 7). In addition,

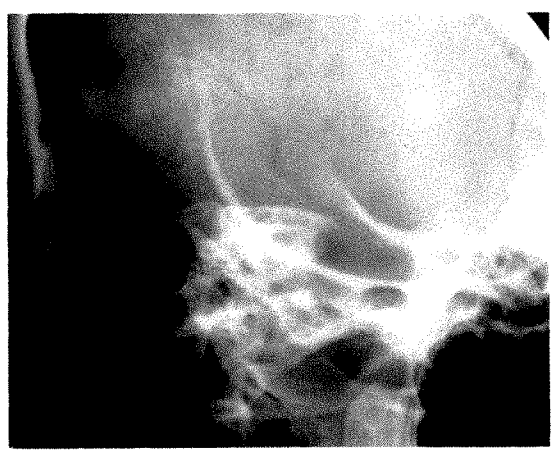


FIG. 5. Similar destruction of left petrous tip from epidermoid tumor.

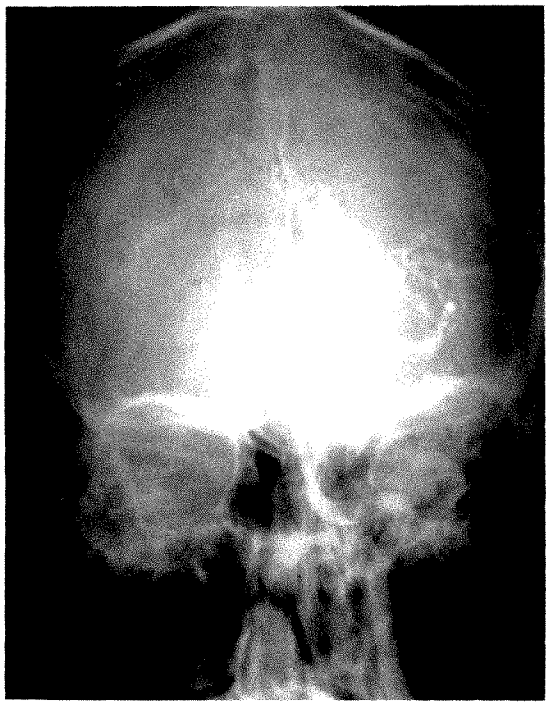


Fig. 6. Medial compression of left carotid siphon and elevation of superior portion of siphon and middle cerebral artery by large parasellar mass.

this patient showed some dislocation of the aqueduct to the opposite side, together with evidence of obstruction of this channel.

Pneumoencephalography was undertaken in a patient whose tumor was at the cerebellopontine angle. Although the lesion

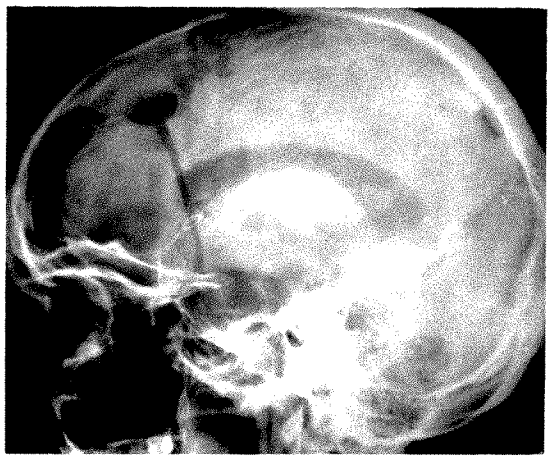


Fig. 7. Elevation and amputation of left temporal horn from recurrent large tumor in middle and posterior fossae.

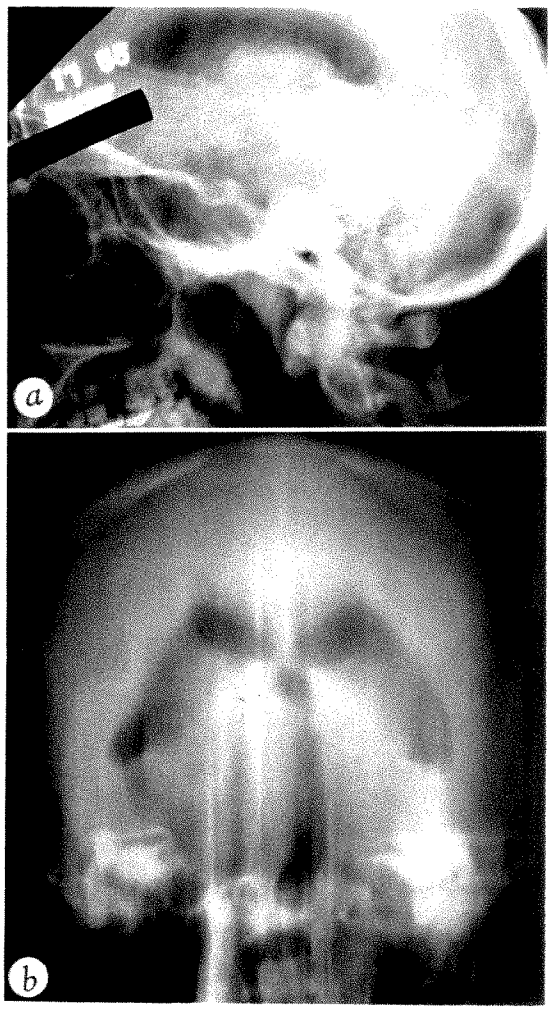


Fig. 8. (*a*) Right lateral pneumoencephalogram. A mass is displacing the aqueduct and fourth ventricle posteriorly. (*b*) Posteroanterior laminagram in same case, showing aqueduct and fourth ventricle displaced to right. Note destruction of the left petrous tip, with a sharp border medial to the internal acoustic meatus.

could be seen in the regular pneumoencephalogram, it was evident by means of laminagraphy that the aqueduct and fourth ventricle were displaced to the opposite side and that the aqueduct was displaced posteriorly by a mass compressing this portion of the brain (Fig. 8, *a* and *b*).

COMMENT

Previous authors have described some roentgenographic features associated with neurofibromas of the trigeminal nerve. In

1955, Jefferson[1] stressed regular but considerable enlargement of the foramen ovale and the foramen rotundum and erosion of the petrous bone as important findings. None of our patients had obvious isolated involvement of the foramen rotundum roentgenographically or at operation. Regarding pneumoencephalography, Lindgren[2] stated that displacement of the homolateral temporal horn was likely to be found in tumors of the fifth cranial nerve confined to the middle cranial fossa, and that extension of the tumor downward into the posterior fossa would produce evidence of hydrocephalus and displacement of the aqueduct and fourth ventricle. Lindgren concluded that carotid arteriography can afford only an approximate idea of that part of the tumor situated in the middle fossa of the skull, and that its usual value is in eliminating the possibility of an aneurysm.

Little mention apparently has been made of the possibility of a soft-tissue mass located in the pterygoid fossa and extending into the posterior pharyngeal soft tissues. Such a mass is common when the gasserian ganglion is involved by a metastatic process. It is important to note, therefore, that the presence of a retropharyngeal soft-tissue mass does not exclude the possibility of neurofibroma of the fifth nerve.

It is evident that the bony changes usually are related to the slow expansion of a mass and not to bony invasion by a neoplastic process, although the tumor in one case of our series grew through the petrous bone and presented in the middle ear and external auditory canal. Therefore, the changes observed are not those usually seen with other lesions in this region, such as chordomas, metastatic tumors, chemodectomas and pyoceles.

In general, these tumors attain a rather considerable size before disabling symptoms are observed. This may be related to the fact that the tumors arise from the nerve sheath and do not directly involve the functioning elements of the nerve until the lesions surround or expand them. This is different from the situation in which the gasserian ganglion is involved by a metastatic tumor. The chief differential diagnostic possibilities to be considered when the lesion is primarily in the middle fossa include aneurysm of the internal carotid artery and parasellar extension of an intrasellar adenoma. In most instances, this differentiation can be made by angiography.

By far the most characteristic single roentgenologic finding appears to be the type of erosion of the tip of the petrous bone. It should not be implied that this is the only pathologic process to produce such a sharp cutoff of the petrous tip medial to the internal acoustic meatus, but it is rather striking that this change was apparent in all 8 cases in which the petrous tip was involved. In most cases of bony involvement by a cerebellopontine angle tumor caused by other lesions, the changes are not so clear cut. When this change in the petrous tip is not present, there is little possibility of distinguishing this neurofibroma from one arising from the eighth nerve, which may not change the petrous bone. If it is remembered that most neurofibromas of the eighth nerve first alter the borders of the internal acoustic meatus, the presence of isolated destruction of the petrous tip might alert one to check carefully for localized changes in the middle fossa as well, in order to recognize a fifth-nerve neurofibroma that may have components in both the middle and posterior fossae. As study of the clinical features in these cases suggests, it is not possible always to make this distinction on a clinical basis. None of our patients showed any erosion of the internal acoustic meatus itself.

Erosion of bony structures within the middle fossa was observed in more than half of our patients. This varied from decalcification around the foramen ovale to extensive destruction of the floor of the middle fossa and the sella turcica. These tumors may cause considerable destruction of the middle fossa without extending very far into the soft tissues below.

Decalcification of the sella turcica from

increased intracranial pressure was encountered in 3 instances. This, of course, also is associated with other growths, particularly those causing obstruction to the ventricular system.

SUMMARY AND CONCLUSIONS

A study was made of the roentgenologic features in 14 cases at the Mayo Clinic in which neurofibromas involved the gasserian ganglion. Primary neurofibromas of the gasserian ganglion occur in the middle or the posterior fossa or both. They are rare and may present a problem in diagnosis because of a paucity of initial symptoms referable to involvement of the fifth cranial nerve.

The most characteristic roentgenologic feature is sharply circumscribed erosion of the petrous tip medial to the internal acoustic meatus. This change occurred in 8 of the 14 cases. Destructive changes in the middle fossa were encountered in more than half of the cases. A soft-tissue mass in the pterygoid fossa and posterior nasopharynx was recognized in 2 instances.

Pneumoencephalography and ventriculography are helpful in confirming the presence and location of the mass, but features pathognomonic of neurofibroma of the fifth cranial nerve were not found in this study.

Carotid angiography is helpful in excluding aneurysms and pituitary fossa tumors and in confirming the presence of a mass in the middle fossa.

Mayo Clinic
Rochester, Minnesota

REFERENCES

1. Jefferson, G. Trigeminal neurinomas with some remarks on malignant invasion of gasserian ganglion. In: Clinical Neurosurgery: Proceedings of the Congress of Neurologic Surgeons. Williams & Wilkins Company, Baltimore, 1955, pp. 11–54.
2. Lindgren, E. Das Röntgenbild bei Tumoren des Ganglion Gasseri. *Acta chir. scandinav.*, 1941, *85*, 181–194.
3. Olive, I., and Svien, H. J. Neurofibromas of fifth cranial nerve. *J. Neurosurg.*, 1957, *14*, 484–505.
4. Schisano, G., and Olivecrona, H. Neurinomas of gasserian ganglion and trigeminal root. *J. Neurosurg.*, 1960, *17*, 306–322.

CEREBRAL ANGIOGRAPHY IN THE DIAGNOSIS OF SUPRASELLAR TUMORS*

By NORMAN E. CHASE, M.D., *and* JUAN M. TAVERAS, M.D.
NEW YORK, NEW YORK

SUPRASELLAR tumors can be demonstrated by cerebral angiography as well as by pneumography. Pneumography is superior to angiography in that it gives an accurate indication of the size and shape of the tumor, as well as its relationship to the third ventricle, the foramen of Monro and the brainstem. Angiography is superior to pneumography in that tumor circulation can be visualized, thus making possible a specific histologic diagnosis, and in that an arterial aneurysm can be distinguished which by pneumography could be mistaken for a neoplasm. The latter is the primary reason why angiography has been growing in popularity in the study of all suprasellar syndromes—particularly in those cases where a meningioma is suspected or when an aneurysm cannot be excluded on clinical grounds. Also by carefully evaluating all of the findings on the angiogram, it is possible to arrive at a surprisingly accurate appreciation of the actual size of the tumor and its relationship to the sella turcica.

It is the purpose of this paper to point out the angiographic signs of suprasellar tumors and to establish criteria for determining size and direction of growth without resorting to pneumography.

PATHOLOGY

The tumors considered here are those which arise from the contents of the sella turcica, such as pituitary adenomas and craniopharyngiomas, or those which arise outside of the sella turcica. The latter group would comprise the suprasellar meningiomas, optic gliomas, epidermoid cysts (included with craniopharyngiomas by some) and aneurysms. Pituitary adenomas are by far the most common tumors in this region and were found in 43 of the 103 cases studied.

Pituitary adenomas may grow exclusively within the sella turcica by enlarging the osseous shell of the sella or they may break through the diaphragma sellae and project upwards. It is the tumor with suprasellar extension that presents with varying degrees of optic nerve and chiasmal compression. The extension of the tumor may be primarily upward but it may be upward and forward or upward and backward. It may also extend laterally.[2,5,11] A knowledge of the direction of growth as well as the extent of the tumor is desirable, particularly if surgery is contemplated.

The second group of tumors in order of frequency were the meningiomas. These may arise from the tuberculum sellae or the planum sphenoidale. They may not necessarily be attached to bone and may arise from the meninges around the cavernous sinus or around the optic nerves.

The third group of tumors studied were the craniopharyngiomas; some undoubtedly originated within the sella turcica and others arose apparently above the sella turcica. In this group are also included the suprasellar epidermoid cysts.

Aneurysms were also studied. These may produce signs of compression of the optic chiasm. In our experience those which caused optic nerve compression usually arose from the internal carotid artery just distal to or at the origin of the ophthalmic artery on the anterior or anteromedial aspect of the supraclinoid portion of the siphon.

Other lesions such as optic gliomas, metastatic pinealomas to the suprasellar region, nonneoplastic cysts and arteriovenous malformations represented but a

* From the Department of Radiology, Columbia Presbyterian Medical Center, New York, New York.
Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

small fraction of the cases analyzed.

Extradural tumors occurring in the region of the sella turcica, such as chordomas and nasopharyngeal carcinomas, will be considered in a subsequent publication.

MATERIAL AND CLASSIFICATION

This study consists of 103 patients, ranging in age from one to seventy-three years (Table I), with suprasellar tumors, who were examined by cerebral angiography. Sixty-two cases were proved by surgical exploration or postmortem examination, the remaining 41 by follow-up studies as well as clinical and roentgenologic evidence of the pathology (Table II). There are 44 males and 59 females in this group.

An exact pathologic diagnosis can be made by angiography only in aneurysms and in those few meningiomas which present a characteristic tumor circulation. Since the vast majority of tumors in our series are not in either of these groups, we have subdivided our cases according to the direction of growth rather than the histology (Fig. 1).

ANGIOGRAPHIC SIGNS OF SUPRASELLAR TUMORS

This problem has been considered by other authors.[6,7,8] The vessels of most significance in the evaluation of tumors in the region of the sella turcica are: the internal carotid artery; the proximal portion of the middle cerebral arteries; the horizontal and proximal ascending portions of the anterior cerebral arteries; the anterior choroidal artery; the posterior communicating and

TABLE I

AGE DISTRIBUTION OF 103 CASES WITH SUPRASELLAR AND PARASELLAR LESIONS

0–10	1
11–20	7
21–30	4
31–40	21
41–50	25
51–60	29
61–70	15
71 and over	1

TABLE II

DIAGNOSIS OF 89 CASES OF SUPRASELLAR AND PARASELLAR LESIONS

A. Pathologically Confirmed Diagnosis	
Pituitary adenomas	20
Meningiomas	14
Craniopharyngiomas	16
Aneurysms	5
Optic gliomas	3
Hypothalamic gliomas	1
Metastatic pinealomas	1
Arteriovenous malformation of interpeduncular space	1
Tortuous carotid arteries compressing optic chiasm	1
Total	62
B. Clinical and Roentgenologic Diagnosis Only	
Pituitary adenomas	23
Meningiomas	3
Aneurysms	1*
Total	27
Total Cases	89†

* One posterior suprasellar mass was considered on clinical and indirect roentgenographic evidence to be a basilar artery aneurysm. However, no vertebral angiography was performed.

† Fourteen cases with extradural tumors are not included.

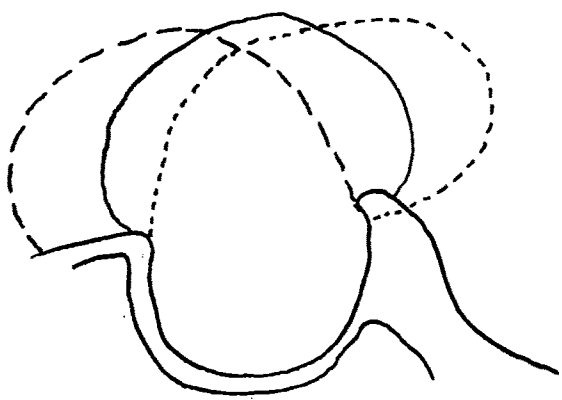


FIG. 1. Diagram of the characteristic directions of growth of suprasellar tumors. The solid line represents upward extension; the dashes represent anterior suprasellar extension; the dotted line represents posterior suprasellar extension. Lateral extension is not illustrated. It is apparent that an enormous tumor may occupy any or all of the above areas.



FIG. 2. Anteroposterior angiogram demonstrating the normal relationship between the anterior portion of the intracavernous segment of the internal carotid artery and the posterior portion (two arrows). The two points are superimposed. Normally the posterior portion is either superimposed upon or slightly medial to the anterior portion. The single arrow points to the anterior clinoid process which is about 0.5 cm. above the anterior end of the intracavernous segment. Note also the normal position of the posterior cerebral artery (p).

posterior cerebral arteries; and the basilar and internal cerebral veins.

I. CHANGES INVOLVING THE INTERNAL CAROTID ARTERY

In order to study the changes involving the internal carotid artery, the intracranial portion of this artery is divided into precavernous, intracavernous and supraclinoid segments.

The intracavernous segment may be displaced laterally. This has been observed pathologically[5,11] and demonstrated on base views of the skull during carotid angiography.[3] However, it is possible to demonstrate lateral displacement on the frontal angiograms as well. Normally, the posterior portion of the intracavernous segment is slightly more medial than the anterior portion (Fig. 2). This is easy to ascertain by following the artery as it comes out from the carotid canal of the petrous bone to determine the posterior portion; by tracing the supraclinoid segment downwards, it is possible to determine the anterior portion. It is sometimes difficult to ascertain whether lateral displacement is present or not, but if the posterior portion is lateral to the anterior portion, lateral displacement is probably present (Fig. 3).

Apparent upward displacement of the intracavernous segment is seen in pituitary adenomas and in craniopharyngiomas accompanied by sellar enlargement. This is due to depression of the floor of the sella turcica, the artery remaining in the same position (Fig. 4). Only tumors with extradural components cause absolute elevation of the intracavernous segment. A pituitary adenoma that extends epidurally may cause absolute upward displacement of this portion of the artery but this is uncommon.

The supraclinoid segment of the internal carotid artery may be: (a) displaced upward (opening of the siphon); (b) displaced downward and backward (closing of the siphon); (c) displaced laterally.

Upward displacement is characteristic of

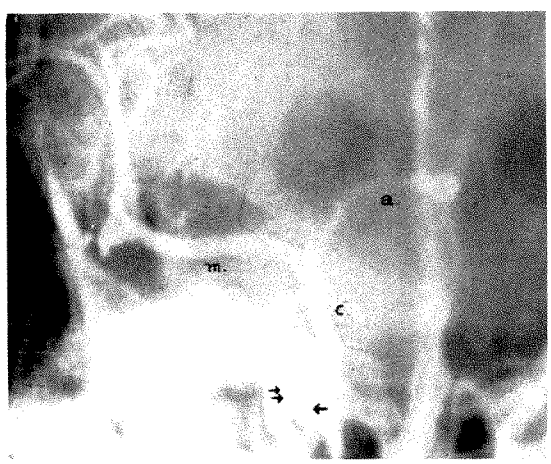


FIG. 3. A histologically verified pituitary adenoma in a fifty-four year old female (unit #137-02-85). The posterior portion of the intracavernous segment of the internal carotid (two arrows) is lateral to the anterior portion (one arrow). Note also the elevation of the carotid bifurcation with the stretching and lateral displacement of the carotid siphon (c) as well as the marked elevation of the anterior cerebral artery (a).

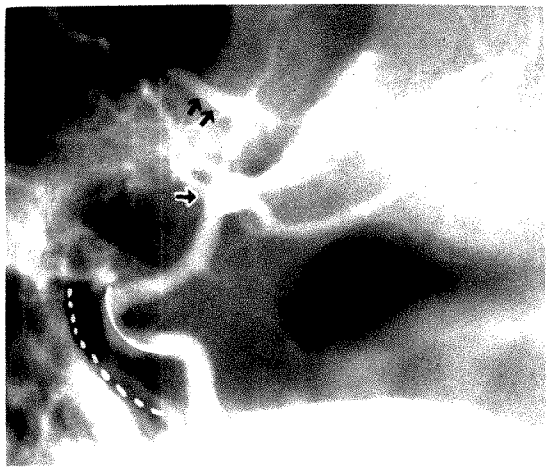


FIG. 4. Large pituitary adenoma (same case as in Figure 3). Dotted line outlines the apparent floor of the eroded sella turcica. Solid line represents the probable position of the floor prior to the erosion by the tumor. It can be seen that the intracavernous segment of the internal carotid artery is not elevated relative to the original floor of the sella turcica. The marked elevation of the carotid bifurcation (one arrow) and the stretching of the carotid siphon as well as the elevation of the proximal anterior portion of the cerebral artery (two arrows) are well demonstrated.

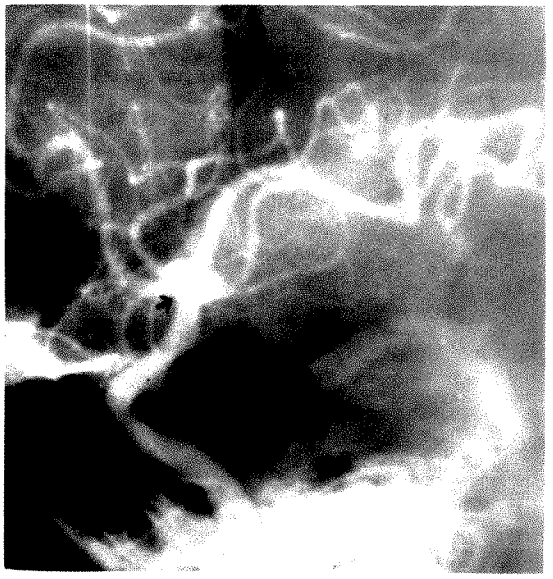


FIG. 5. Large pituitary adenoma in a fifty-two year old male (unit #30-00-70). There are marked elevation of the carotid bifurcation (arrow) and opening of the carotid siphon. The apparent elevation of the middle cerebral artery is related to the marked elevation of the bifurcation.

tumors with intrasellar origins such as pituitary adenomas (Fig. 5). Craniopharyngiomas do not displace the supraclinoid segment upward as frequently (Fig. 6, *A* and *B*), and may indicate in these cases that their origin is primarily extrasellar. Craniopharyngiomas may also originate within the sella turcica but they extend upward

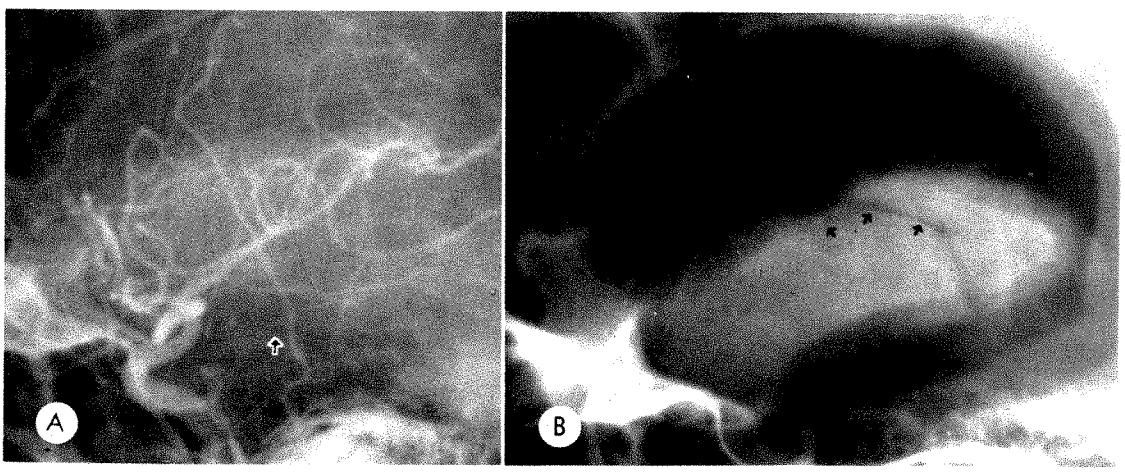


FIG. 6. Intraventricular craniopharyngioma in a thirty-two year old female (unit #133-01-22) who was admitted to the hospital with symptoms of chiasmal compression suggestive of a suprasellar mass. (*A*) The carotid siphon is normal in appearance. No vascular displacement is evident. The arrow points to a posterior branch of the middle meningeal artery not to be confused with a stretched anterior choroidal artery. (*B*) Ventriculogram showing obliteration of entire third ventricle by an enormous intraventricular craniopharyngioma. Arrows outline limits of tumor.

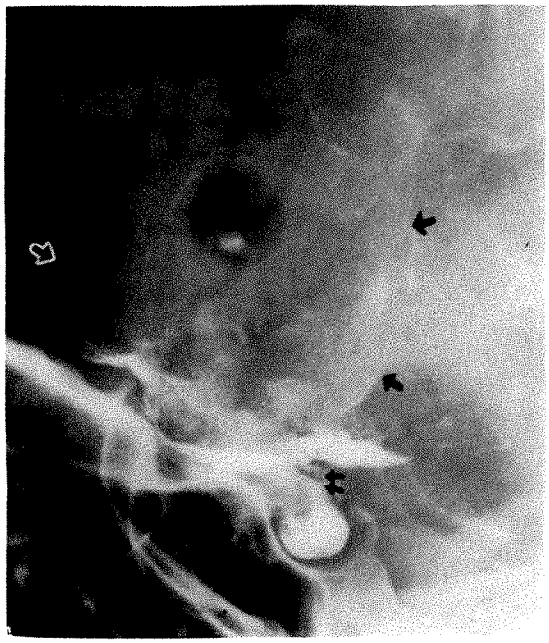


FIG. 7. Surgically verified craniopharyngioma in a seven year old female (unit #16-99-83). Thorotrast has been introduced into the suprasellar cyst (outlined by arrows). A narrow neck (two arrows) is seen connecting the large suprasellar portion with the small intrasellar portion. (Courtesy of Dr. John Scarff, New York City.)



FIG. 8. Histologically proved anterior suprasellar craniopharyngioma in a forty-five year old male (unit #147-40-44). There are closing of the carotid siphon and marked elevation of the anterior cerebral artery (arrows).

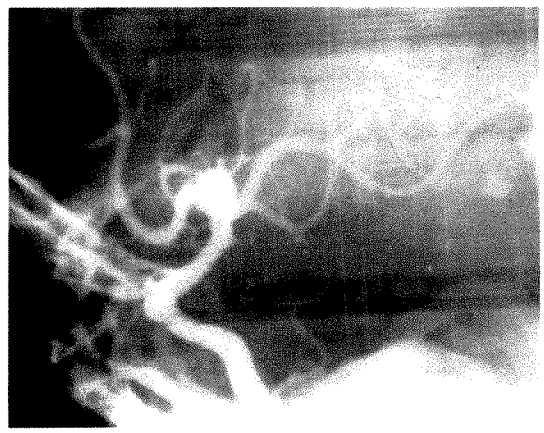


FIG. 9. Posterior suprasellar pituitary adenoma in a thirty-eight year old male (unit #133-03-05). The carotid siphon is open. The proximal portion of the anterior cerebral artery is not displaced upward. Parasellar (infratemporal) extension is also present (see Figures 11 and 17); the middle cerebral artery is elevated.

through the diaphragma sellae as a thin stalk which then mushrooms into a large cyst. In these cases one cannot expect elevation of the supraclinoid segment of the siphon (Fig. 7). This mode of extension is rare in pituitary adenomas.

Tumors in the anterior suprasellar region tend to produce downward and backward displacement of the siphon. This is also true in some pituitary adenomas with forward extension (Fig. 8).

Tumors in the posterior suprasellar region tend to open the siphon (Fig. 9); however, angiograms may show a normal appearance (Fig. 10*A*).

Lateral displacement of the supraclinoid segment of the internal carotid artery is almost always present when tumors lie medially to the vessel. This is true in most cases of pituitary adenomas as well as in most of the other suprasellar tumors (Fig. 3). Medial displacement may be encountered in cases of pituitary adenomas that have extended laterally (Fig. 11). Stretching of the supraclinoid segment and elevation of the bifurcation of the internal carotid artery are fairly common findings in suprasellar tumors, in general.

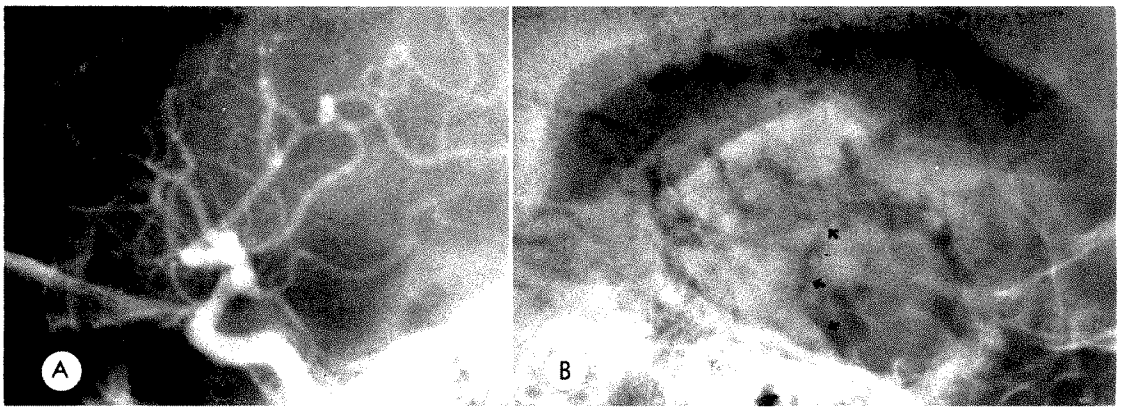


Fig. 10. Posterior suprasellar pituitary adenoma in a sixty year old female. (*A*) Lateral arteriogram shows normal siphon and no definite vascular displacements. (*B*) Lateral pneumoencephalogram demonstrates the extensive tumor mass (arrows).

II. Changes Involving the Anterior Cerebral Artery

The horizontal portion of the anterior cerebral artery is usually displaced upward in tumors that are either directly suprasellar or anterior suprasellar in location (Fig. 3 and 4). In posterior suprasellar tumors the anterior cerebral artery will not be affected even by large tumors (Fig. 6, *A* and *B*; 9; 10, *A* and *B*; and 11). It is often extremely difficult to determine whether the horizontal portion of the anterior cerebral artery is or is not displaced upward, particularly in view of the fact that some degree of elevation may be found in normal individuals and even greater apparent elevation may be seen in individuals with vascular tortuosity (Fig. 12). This apparent elevation is frequently seen in children.[10] The degree of elevation of the anterior cerebral artery is usually determined on the frontal angiograms but roentgenograms with increased angulation showing the orbits may be needed as well as the straight anteroposterior ones. A more reliable sign may be seen on the lateral angiogram (Fig. 4 and 8). Normally, the curve is convex downwards, immediately distal to the siphon. In the presence of anterior suprasellar tumor, this convexity may be flattened or a concavity may be present (Fig. 4, 8 and 13). Posterior displacement of the anterior cerebral artery may occur in the presence of large anterior suprasellar tumors and is usually associated with upward displacement of the vessel.

III. Changes in the Middle Cerebral Arteries

Suprasellar tumors may elevate the proximal portion of the middle cerebral arteries;

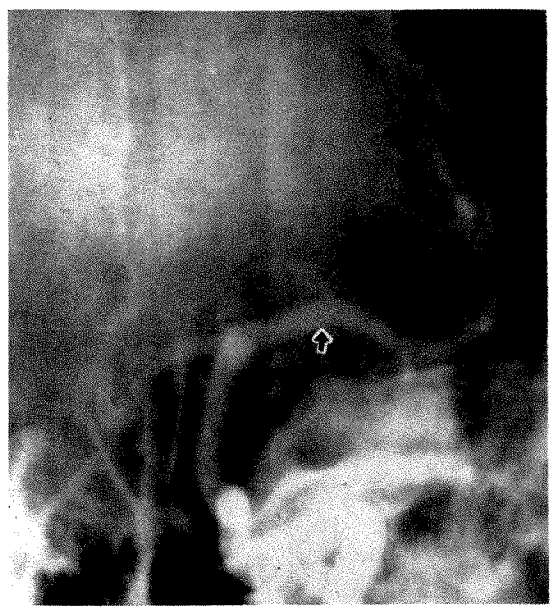


Fig. 11. Large posterior suprasellar pituitary adenoma with lateral extension (same case as in Figure 9). The elevation of the middle cerebral artery (arrow) is greater than the elevation of the carotid bifurcation. The carotid siphon is stretched and medially displaced. No elevation of the anterior cerebral artery is present. (Compare with Figure 3.)

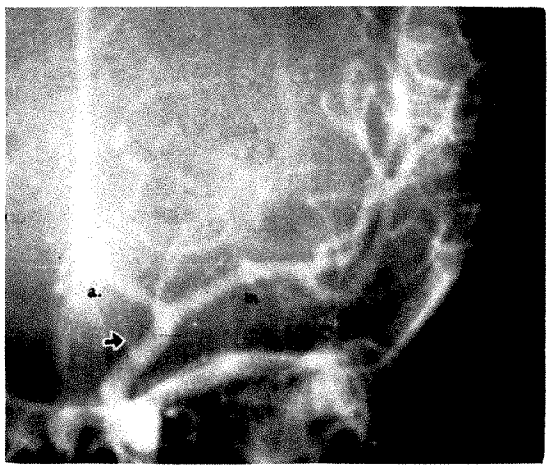


FIG. 12. Patient with no clinical evidence of chiasmal compression or endocrine changes. There is apparent elevation of the horizontal portion of the anterior cerebral artery (a). This is a frequent normal finding (see text). The posterior communicating artery is in a normal position (arrow).

this elevation is usually an indication of the upward displacement of the carotid bifurcation and only occasionally is it due to infratemporal extension of suprasellar tumors (Fig. 3 and 11). Lateral displacement of the ascending branches of the middle cerebral arteries may be found in cases of massive suprasellar tumors which produce ventricular dilatation secondary to obstruction of the foramen of Monro.

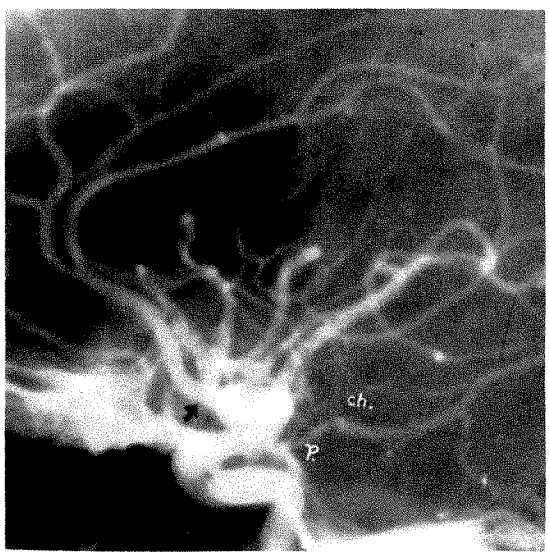


FIG. 13. Normal lateral arteriogram demonstrating the downward convexity usually seen in the proximal portion of the anterior cerebral artery (arrow). Compare with Figures 4, 8 and 17. The anterior choroidal (ch) and posterior communicating (p) arteries are in normal positions.

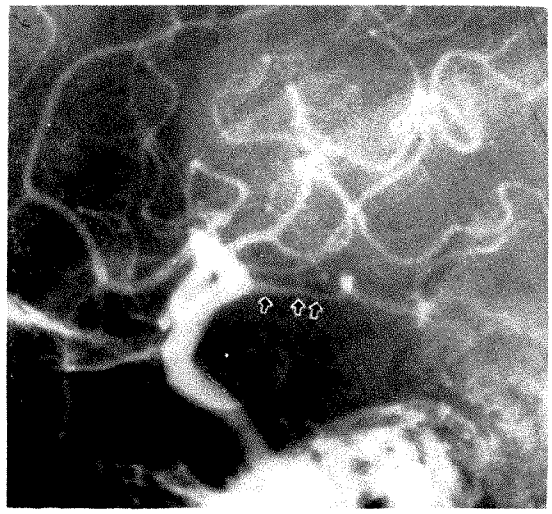


FIG. 14. A histologically verified pituitary adenoma in a sixty-two year old female (unit #24-98-13). There are elevation and forward displacement of the carotid bifurcation and elevation of the posterior communicating and anterior portion of the posterior cerebral arteries (arrows).

IV. CHANGES IN THE POSTERIOR COMMUNICATING AND POSTERIOR CEREBRAL ARTERIES

The posterior communicating and posterior cerebral arteries may be deflected superiorly and laterally by posterior suprasellar tumors (Fig. 14 and 15, *A*, *B* and *C*). Upward displacement of the posterior communicating and proximal posterior cerebral arteries may be very difficult to evaluate. Lateral displacement, as seen on the frontal angiogram, is easier to ascertain (Fig. 15*A*). Normally, the posterior communicating artery extends posteriorly and medially from its origin in the internal carotid artery. When laterally displaced, the posterior portion is either superimposed upon or lateral to its anterior portion. The presence of a posterior suprasellar mass can often be determined on angiograms only by the lateral displacement of the posterior communicating, posterior cerebral and anterior choroidal arteries. If these arteries do not

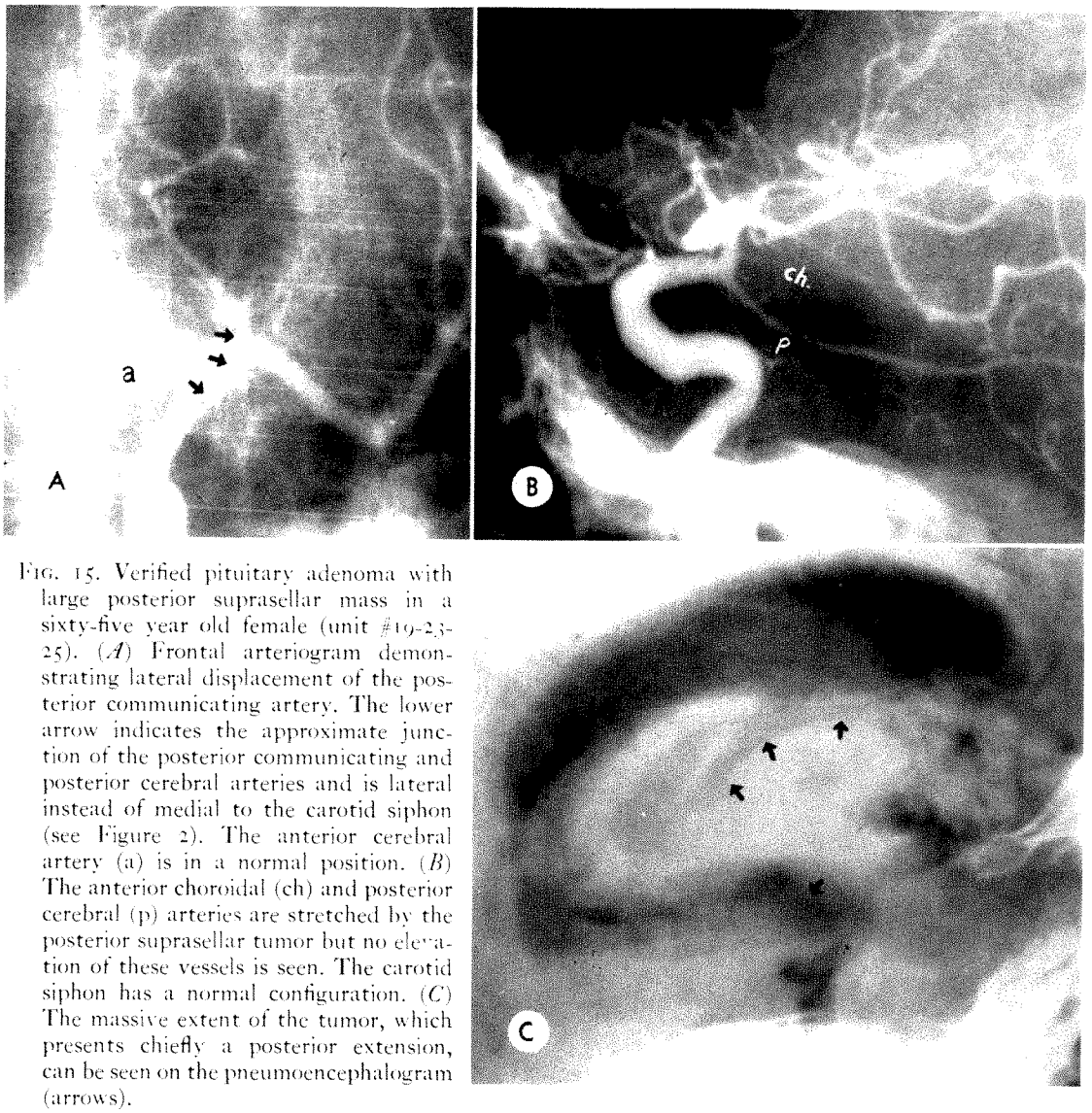


Fig. 15. Verified pituitary adenoma with large posterior suprasellar mass in a sixty-five year old female (unit #19-23-25). (*A*) Frontal arteriogram demonstrating lateral displacement of the posterior communicating artery. The lower arrow indicates the approximate junction of the posterior communicating and posterior cerebral arteries and is lateral instead of medial to the carotid siphon (see Figure 2). The anterior cerebral artery (a) is in a normal position. (*B*) The anterior choroidal (ch) and posterior cerebral (p) arteries are stretched by the posterior suprasellar tumor but no elevation of these vessels is seen. The carotid siphon has a normal configuration. (*C*) The massive extent of the tumor, which presents chiefly a posterior extension, can be seen on the pneumoencephalogram (arrows).

fill well, the tumor may be overlooked (Fig 6, *A* and *B*; and 10, *A* and *B*).

V. Changes in the Anterior Choroidal Artery

The origin of the anterior choroidal artery is in the internal carotid artery, 1 or 2 mm. distal to the origin of the posterior communicating artery. As was true with the posterior communicating and posterior cerebral arteries, posteriorly placed suprasellar masses may cause upward and lateral displacement of the anterior choroidal arteries. Upward displacement is often very difficult to evaluate unless it is massive. Lateral displacement may be determined on the frontal angiograms. This is usually present when the most medial portion of the artery (the portion that passes about the uncus) is superimposed on or lateral to the internal carotid artery. Normally, this segment projects medially to the siphon (Fig. 16 and 17). Inferior and lateral displacement of the anterior choroidal and posterior communicating arteries may occur with posterior suprasellar tumors that

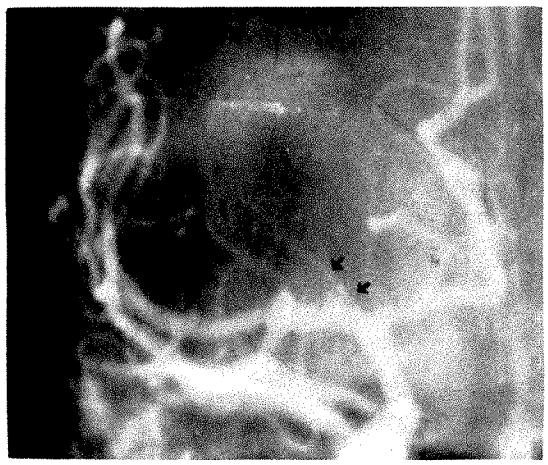


FIG. 16. Posterior suprasellar craniopharyngioma in a fifty-seven year old male (unit #148-36-40). There is considerable lateral displacement of the anterior choroidal artery (arrows).

are extrasellar in origin, particularly in cases of tumors originating in the third ventricle.

VI. CHANGES IN THE INTERNAL CEREBRAL VEIN AND THE BASILAR VEIN

The internal cerebral vein and the basilar vein are seldom noted to be affected by suprasellar tumors. Tumors which extend laterally may cause elevation of the basilar vein (Fig. 18). There were 8 such cases in our series. Massive suprasellar tumors may elevate the anterior portion of the internal cerebral vein (Fig. 19).

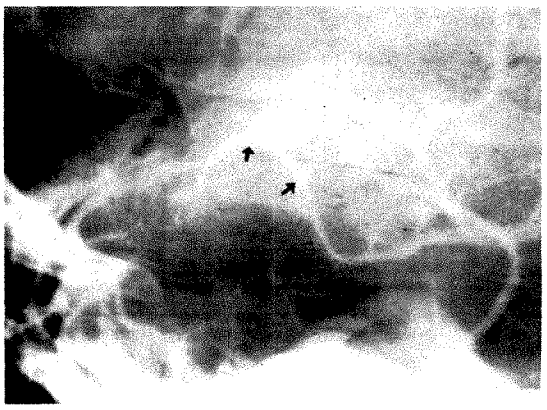


FIG. 18. Same case as in Figures 9 and 11. Marked elevation of the basilar vein can be seen with infratemporal extension of a pituitary adenoma (arrows).

COMMENT

In addition to aneurysms, pituitary adenomas can be diagnosed accurately by angiography. It is apparent that all pituitary adenomas begin as intrasellar tumors. The degree of erosion and expansion of the sella turcica depends primarily on the strength and completeness of the diaphragma sellae. Since the medial dural wall of the cavernous sinus is relatively thin and flexible, an expanding process in the sella

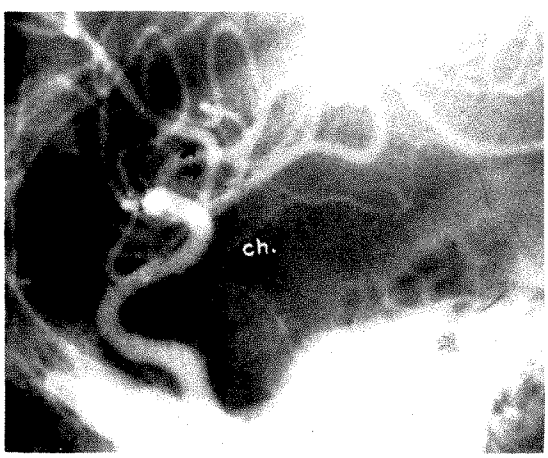


FIG. 17. A histologically verified large suprasellar pituitary adenoma in a forty year old male (unit #20-73-07). There is elevation of the carotid siphon as well as elevation of the anterior cerebral artery (arrows) and anterior choroidal artery (ch).

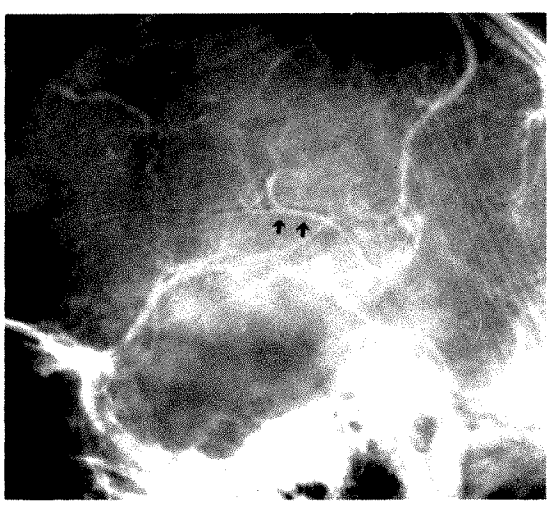


FIG. 19. Enormous pituitary adenoma in a fifty-one year old male (unit #29-98-89) causing elevation of the internal cerebral vein (arrows).

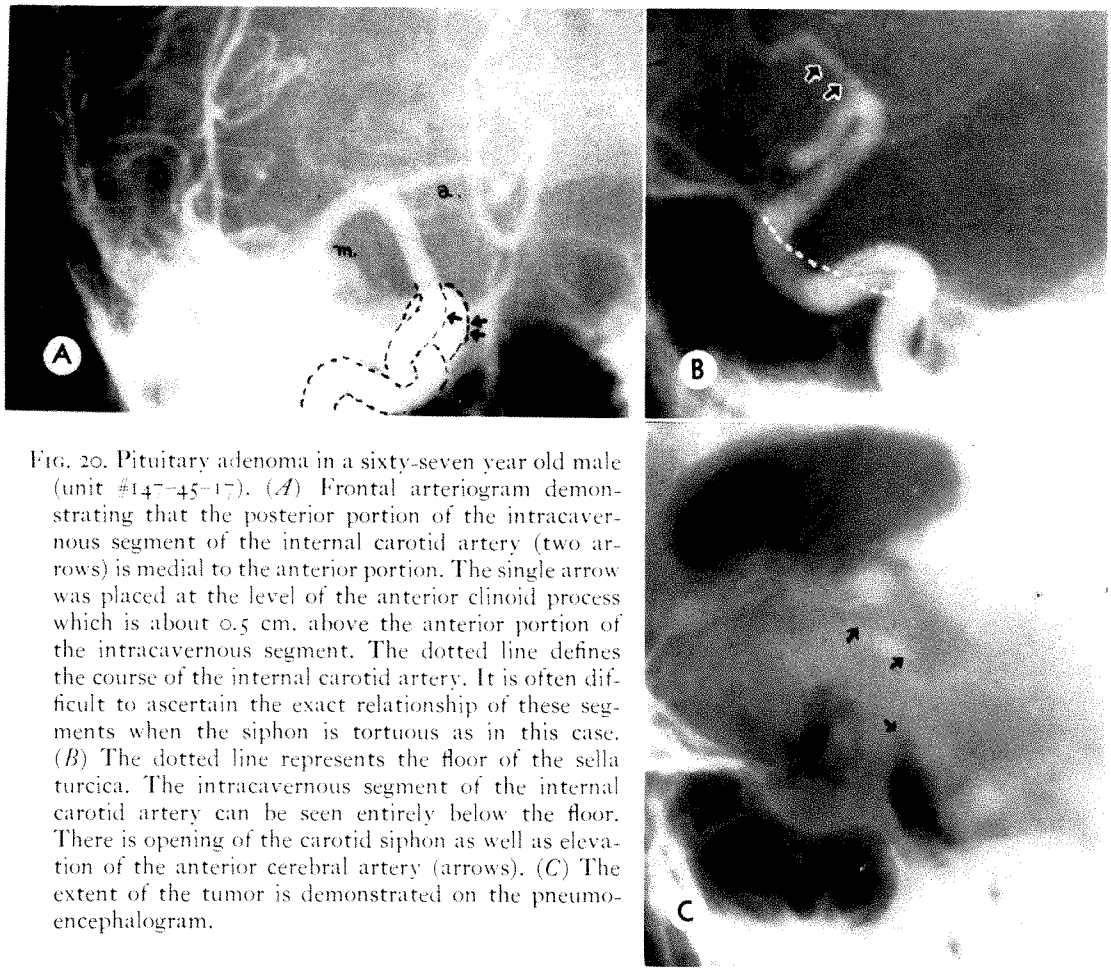


FIG. 20. Pituitary adenoma in a sixty-seven year old male (unit #147-45-17). (*A*) Frontal arteriogram demonstrating that the posterior portion of the intracavernous segment of the internal carotid artery (two arrows) is medial to the anterior portion. The single arrow was placed at the level of the anterior clinoid process which is about 0.5 cm. above the anterior portion of the intracavernous segment. The dotted line defines the course of the internal carotid artery. It is often difficult to ascertain the exact relationship of these segments when the siphon is tortuous as in this case. (*B*) The dotted line represents the floor of the sella turcica. The intracavernous segment of the internal carotid artery can be seen entirely below the floor. There is opening of the carotid siphon as well as elevation of the anterior cerebral artery (arrows). (*C*) The extent of the tumor is demonstrated on the pneumoencephalogram.

turcica will usually affect the cavernous sinus, displacing it laterally and expanding the floor.[5,11] This can be demonstrated by angiography except in those cases in which the sella turcica is shallow and associated with extensive pneumatization of the sphenoid sinus (Fig. 20, *A*, *B* and *C*). Individuals with this anatomic variant often have an incomplete or a weak diaphragma sellae. Also in these instances, most of the cavernous sinus lies below the level of the floor of the sella turcica which is usually normal in size. Because the weak diaphragma sellae offers little resistance to the upward expansion of the tumor, lateral displacement of the intracavernous portion of the internal carotid artery does not occur.[4] There were 4 cases of this type in this series.

Small tumors of the tuberculum sellae are difficult to detect angiographically since the anterior cerebral arteries, the only vessels that might be displaced by these tumors, are not affected. One case of a small meningioma arising from the optic nerve sheath showed no angiographic abnormalities. Indeed, neither are direct changes seen on pneumoencephalograms with the exception of the distortion of the parolfactory sulci.

It should be mentioned that some craniopharyngiomas or "suprasellar gliomas" may arise in the hypothalamus, and in these cases no specific vascular displacement may be seen other than lateral displacement and occasionally downward displacement of the anterior choroidal and, or

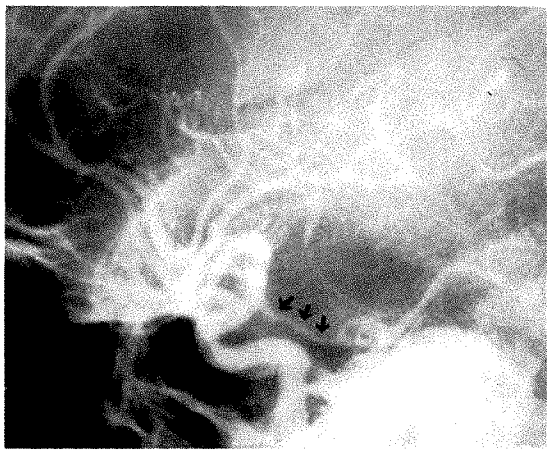


FIG. 21. Intraventricular craniopharyngioma in a forty year old female (unit #21-08-25). The arteriogram demonstrates downward displacement of the posterior communicating and posterior cerebral artery (arrows). Ventriculograms showed marked ventricular dilatation and a large mass filling the third ventricle almost completely. At operation the tumor proved to be an intraventricular craniopharyngioma.

posterior communicating arteries (Fig. 21).

Posterior suprasellar tumors with no intrasellar component were the most difficult to diagnose by angiography. These tumors, even though relatively large, may not displace the anterior choroidal, the posterior communicating and posterior cerebral arteries; and even if these vessels are visualized, the tumors will escape detection (Fig. 6, *A* and *B*).

In 54 of the 103 cases reviewed, pneumography as well as angiography was performed. Pneumography was felt to be superior in 48 per cent of these cases; both procedures were considered equal in 27 per cent, and arteriography was considered superior in 25 per cent.

SUMMARY

One hundred and three cases of suprasellar tumors with angiographic studies have been reviewed. One-half of these had pneumography as well. In two-thirds of the cases the diagnosis was confirmed at surgery or autopsy. In the majority of cases the diagnosis can be made by angiography alone.

The vessels of most significance in the evaluation of these tumors are the precavernous, intracavernous and supraclinoid segments of the internal carotid arteries; the anterior choroidal arteries; the posterior communicating and posterior cerebral arteries; and the proximal portion of the middle cerebral and the anterior cerebral arteries. The basilar and internal cerebral veins have occasionally been of value.

In most instances angiography has equaled pneumography in establishing the diagnosis of suprasellar tumors. In the majority of cases of pituitary adenomas, angiography has been superior to pneumography in determining the type of tumor. The lateral displacement of the intracavernous portion of the internal carotid artery is fairly characteristic of pituitary adenoma as against other types of suprasellar tumors not originating within the sella turcica. *Anterior suprasellar* masses may be distinguished by the elevation of the anterior cerebral arteries and the posterior displacement of the supraclinoid segment of the internal carotid arteries (closing of the siphon). *True suprasellar* tumors usually produce elevation and lateral displacement of the carotid siphon, elevation of the carotid bifurcation (opening of the carotid siphon), and, occasionally, lateral displacement of the anterior choroidal and/or posterior communicating arteries. *Posterior suprasellar* tumors are the most difficult to diagnose angiographically. When diagnosable, they have caused lateral displacement and elevation of the anterior choroidal and posterior communicating and posterior cerebral arteries. It has not been possible in most instances to determine the posterior extent of these tumors by angiography alone. Tumors which extend laterally may lift the temporal lobe and thus produce elevation and lateral displacement of the middle cerebral artery and basilar vein. They may also cause medial displacement and elevation of the supraclinoid segment of the internal carotid artery.

Aneurysms presenting with chiasmal syndromes have been found to originate

primarily from the internal carotid artery at or near the junction of the ophthalmic artery and may not be differentiated by pneumography from tuberculum sellae or planum sphenoidale tumors. It is usually necessary to perform bilateral angiography when studying suprasellar tumors in order to rule out an aneurysm, for there may be nonfilling of the aneurysm from one side even on cross compression.

Bilateral angiography is also of value for a comparison of both sides in determining the presence of small tumors causing relatively subtle changes in the vessels of the circle of Willis.

Norman Chase, M.D.
Department of Radiology
Columbia Presbyterian Medical Center
622 West 168th Street
New York 32, New York

REFERENCES

1. Biggart, J. H., and Dott, N. M. Pituitary tumors; their classification and treatment. *Brit. Med. J.*, 1936, *2*, 1206–1208.
2. Cushing, H. Chiasmal syndrome of primary optic atrophy and bitemporal field defects in adults with normal sella turcica. *Arch. Ophth.*, 1930, *3*, 505–551; 704–735.
3. Ecker, A., and Riemenschneider, P. A. The Angiographic Localization of Intracranial Masses. Charles C Thomas, Publisher, Springfield, Ill., 1953.
4. Frazier, C. H. Review, clinical and pathological, of parahypophyseal lesions. *Surg., Gynec. & Obst.*, 1936, *62*, 158–166.
5. Jefferson, G. Extrasellar extensions of pituitary adenomas; President's address. *Proc. Roy. Soc. Med.*, 1940, *33*, 433–458.
6. Joy, H. H., Ecker, A. and Riemenschneider, P. A. Role of cerebral angiography in ophthalmology. *Am. J. Ophth.*, 1954, *37*, 55–68.
7. Lindgren, E. Röntgenologie. Volume II. Handbuch der Neurochirurgie. Springer-Verlag, Berlin, 1954.
8. Money, R. A., and Vanderfield, G. K. Angiography in management of intra- and suprasellar tumours. *J. Neurosurg.*, 1955, *12*, 203–215.
9. Schwartz, C. W. Tumors of hypophysis cerebri; from a roentgenologic viewpoint. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 548–570.
10. Taveras, J. M., and Poser, C. M. Roentgenologic aspects of cerebral angiography in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 371–391.
11. Trumble, H. C. Pituitary tumours. *Brit. J. Surg.*, 1951, *39*, 7–24.

COMPARISON OF CEREBRAL ANGIOGRAPHIC CONTRAST MEDIA BY THEIR CIRCULATORY EFFECTS*

AN EXPERIMENTAL STUDY

By HARRY W. FISCHER, M.D.,† *and* JOHN W. ECKSTEIN, M.D.‡

CEREBRAL angiography has never been a completely innocuous procedure. Despite improvements in techniques and in contrast materials, a significant incidence of complications yet remains. These complications are transient and mild, or permanent and severe. To know whether the complications following cerebral angiography are due to the contrast material and the injection technique or to the progression of the investigated disease process is difficult. Most intracranial lesions in which cerebral angiography is indicated are capable of exhibiting sudden worsening of symptoms and signs at any time in their course. Adequate clinical comparison of contrast media requires large groups of patients with identical lesions and clinical courses, so that differences in postangiographic status can be correctly ascribed to the contrast material. Although clinical studies may be planned with great care, the lack of sufficient numbers of comparable patients and the great unpredictable variability of disease processes do not allow proper experimental control.

Animal experimentation, therefore, occupies an important place in the evaluation of the contrast media in cerebral angiography. We should say that any satisfactory contrast material must meet two requirements. It should be of a physicochemical nature to supply the information desired, and it should disturb the normal working of the body little or not at all.

We have concerned ourselves with the second requirement, the effect of the contrast materials on the body, but the first requirement, which is largely based on radiopacity, is related to our problem.

Evaluation of cerebral angiographic contrast agents by the degree of response of organs, or the body as a whole, has been obtained in several ways (Fig. 1). The immediate and residual neurologic status of the subject can be observed and expressions of distress, involuntary movements, the severity and type of convulsions measured. Electroencephalography, electrocardiography, recording of blood pressures, cerebral spinal fluid pressure and cerebral blood flow and respiratory measurements give more quantitative and delicate information on the less apparent effects of cerebral angiography. Testing of altered permeability of the blood-brain-barrier and histologic studies of the organs have revealed cytotoxic effects.

We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory CO_2 concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material. Alterations of this type in part were first noted by Weatherall,[26] and later by Broman and Olsson,[4] Foltz *et al.*[9] Bassett and Rogers,[1] Schmidt,[22] and Lindner *et al.*[19] Greitz[10] studied the same type of phenomena in man, and Lindgren and Törnell[16,17] recently made more detailed studies in cats. Several reasons prompted the preference of this to previous methods. The present study allowed an evaluation based on amounts and concentrations of material comparable

* From the Departments of Radiology† and Internal Medicine,‡ State University of Iowa, College of Medicine, Iowa City, Iowa.

This investigation was aided by research grant H4830 of the National Institute of Health of the United States Public Health Service, and a grant from the Iowa Heart Association. Dr. Eckstein is an Established Investigator of the American Heart Association.

Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

to those used in clinical cerebral angiography, whereas most of the previous studies were based upon injection of amounts so excessive as to be unrelated to clinical usage. The measured vital signs are of well understood importance to normal functioning. Any disturbance of these signs by a diagnostic roentgenologic procedure is therefore worthy of consideration. The present method of evaluation was performed on the intact animal in a fashion very similar to clinical angiography, and can be applied to the human patient without difficulty. The measurement of change is quantitative, graphic and nonsubjective and does not require death of the animal.

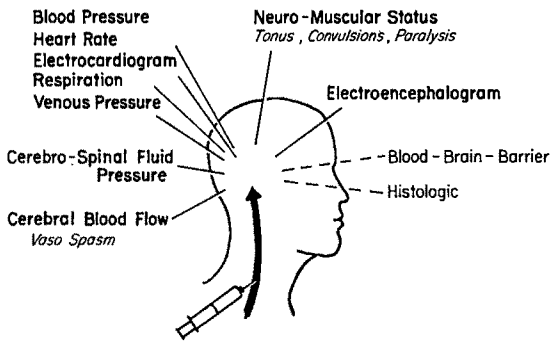


Fig. 1. Contrast media for cerebral angiography have been compared experimentally for their toxic effects by one or more of the above illustrated methods of detection.

DESIGN OF EXPERIMENTS

The injections were given in two separate patterns:

Pattern I. Originally, two contrast materials were compared in a single dog by giving a series of 5 or 6 injections of which injections 1, 3 and 5 were of contrast material A, and injections 2, 4 and 6 were of contrast material B. Equal amounts of the two contrast materials were compared. The doses injected were 0.14 to 0.42 cc. per kg. of body weight of 50 per cent solutions of hypaque, urokon, diodrast, miokon and cardiografin. Only one dose level was tested per dog. Dissatisfaction with this design resulted because of the wide range of response of individual dogs, and because we reasoned that the contrast agents, although of equal chemical concentration and approximately equal tonicity, were not being compared on an equal basis as to radiopacity.

Pattern II. Three contrast materials, hypaque, urokon, and cardiografin, were compared in each of 6 dogs. Two dose levels were tested, 0.15 and 0.30 cc. per kg. of body weight. A Latin square[8] was used to eliminate variability among dogs by randomization of the order of injection. Two doses were selected in which a measurable response was anticipated. Letters A and B of the square were assigned to 0.15 cc. and 0.30 cc./kg. amounts of 50 per cent hypaque, C and D to like amounts of 61 per cent cardiografin, and E and F to like amounts of 45.5 per cent urokon. Hypaque was chosen as a standard because of its wide clinical use. Urokon and cardiografin concentrations were adjusted to equate their iodine contents to that of 50 per cent hypaque.

CONDITIONS PERTINENT TO BOTH PATTERNS OF INJECTION

All experiments were performed on adult dogs, 10 and 20 kg., under intravenous nembutal solution sufficient to induce and maintain surgical anesthesia. Respiration was maintained at a constant rate and volume, beginning immediately after anesthetization by a Harvard fixed volume respirator-pump connected to an inlying intratracheal tube with inflated cuff. Respiration of constant rate and volume is not normal for clinical angiography, but we thought it appropriate to eliminate variability in respiration. Other responses could then be better analyzed. With the dog in the recumbent position, the right common carotid artery was exposed in the lower neck. Just prior to each puncture and injection the carotid was proximally clamped, to minimize inconstancy of mixing of the cerebrally flowing contrast material by blood. All injections were made by the same operator individually puncturing the artery with a 20-gauge needle. Injections

were from a 10 cc. syringe, by manual pressure in Pattern I trials, and by a Shipps injector in Pattern II. The use of the pressure injector required an interconnecting polyethylene tube between needle and syringe. The Shipps injector allowed equating injection times of contrast solutions of different viscosity. Successive injections were made at 15 to 25 minute intervals to allow return of conditions to a pre-injection level insofar as possible.

A Cournand needle was placed in the right femoral artery. A cardiac catheter was inserted into the vena cava through the right femoral vein. Mean arterial blood pressure was determined by electrical integration of the Statham strain gauge output and the pressure response was expressed in mm. Hg. The venous pressure, also measured by a Statham strain gauge, was expressed as the peak response in mm. Hg.

Electrocardiograms were obtained, using the standard leads. The heart rate reduction was expressed in beats per minute for an arbitrary ten second period after injection. This length of time was sufficient to include the major periods of asystole, pronounced bradycardia and arrhythmia. A Beckman medical gas analyzer (Spinco Model LB-1) was connected to the intratracheal tube for the CO_2 measurements. Ventilation was adjusted so that the animal was well oxygenated, with his end-expiratory CO_2 concentration about 4 per cent. Changes in CO_2 concentration were then expressed in arbitrary units. The Sanborn direct writing oscillograph allowed simultaneous tracings of all measurements.

In the Latin square experiment, the responses to each dose level of each contrast solution were averaged. When individual responses were expressed as decrease in mm. Hg or decrease in heart rate, *et cetera*, the baseline level was approximately the same before each response. Responses were expressed as decreases in mm. Hg or beats per minute from baseline, not as percentage decrease.

The contrast materials were taken from ordinary commercial vials, not previously exposed to air or light. Bubbles and small clots were avoided.

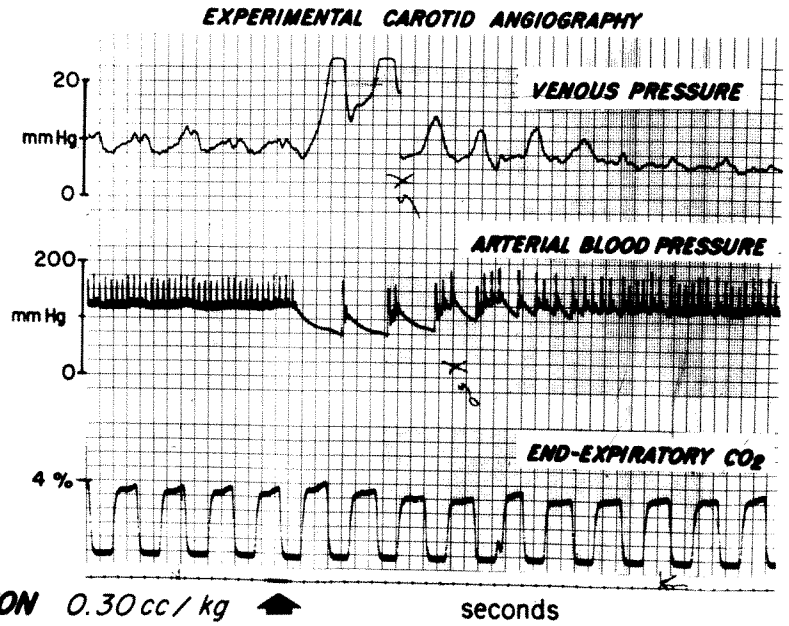


FIG. 2. A typical recording of the cardiovascular disturbances following the intracarotid injection of a contrast solution in the dog, in this case 0.30 cc./kg. of 45.5 per cent urokon. The periods of asystole, bradycardia, arterial hypotension, elevation of venous pressure, and decrease in end-expiratory CO_2 content are to be noted. The period of injection is indicated by the black bar on the time line.

Table I

		Cardiografin	Hypaque	Urokon
Arterial Blood Pressure Fall (mm. Hg)				
0.15 cc./kg.	Range	0–20.0	0–60.0	5.0–130.0
	Average	5.0	18.3	53.0
0.30 cc./kg.	Range	5.0–75.0	5.0–95.0	60.0–120.0
	Average	26.7	56.0	91.0
Duration of Major Blood Pressure Abnormality (seconds)				
0.15 cc./kg.	Range	0–14.0	0–18.0	2.0–31.0
	Average	3.3	5.3	10.8
0.30 cc./kg.	Range	0–15.0	2.0–20.0	2.0–21.0
	Average	5.0	8.0	12.5
Decrease in Heart Rate in the 10 Seconds Post Injection (beats per minute)				
0.15 cc./kg.	Range	0–78.0	6.0–132.0	18.0–144.0
	Average	16.8	36.0	78.0
0.30 cc./kg.	Range	12.0–72.0	24.0–156.0	48.0–192.0
	Average	34.0	66.0	125.0
Peak Rise in Venous Pressure (mm. Hg)				
0.15 cc./kg.	Range	0–1.5	0.5–3.5	0– 5.0
	Average	0.7	1.3	3.0
0.30 cc./kg.	Range	0.5–4.0	0.5–4.0	2.5–11.0
	Average	1.4	3.1	6.3
Reduction in End-Expiratory CO_2 Concentration (arbitrary units)				
0.15 cc./kg.	Range	0.5–2.0	0.5–3.0	1.0–7.0
	Average	1.0	1.5	5.0
0.30 cc./kg.	Range	0.0–5.0	1.0–9.0	3.0–9.0
	Average	1.5	3.7	6.2

RESULTS

The most complete information was obtained from the Pattern II injections, comparing the animals' responses to cardiografin, hypaque, and urokon of approximately equal iodine content. Similar, but on the whole less precise, information was obtained from the Pattern I injections, in which 50 per cent solutions of cardiografin, diodrast, hypaque, miokon and urokon were injected.

The responses depended upon the contrast material and its dose. In almost every instance a greater disturbance was produced by the dose of 0.3 cc./kg. than by the 0.15 cc./kg. dose of each of the three contrast agents.

A typical positive response consisted of pronounced slowing of the heart and/or a brief period of asystole. The response began before the completion of the injection or within two seconds after completion. The electrocardiographic changes were those usually associated with vagal stimulation of the heart. Pronounced bradycardia and short periods of asystole were the most common forms of arrhythmia. Atrial and ventricular premature beats were seen rarely. Other focal arrhythmias were not seen, nor were there instances of atrial or ventricular fibrillation. With slowing or standstill of the heart, a fall of arterial blood pressure occurred, the magnitude of which correlated with the severity and duration of the changes in heart rate. Occurring with the bradycardia, asystoles, and hypotension was an elevation of venous pressure. End-expiratory CO_2 concentration decreased simultaneously. An explanation of the end-expiratory CO_2 concentra-

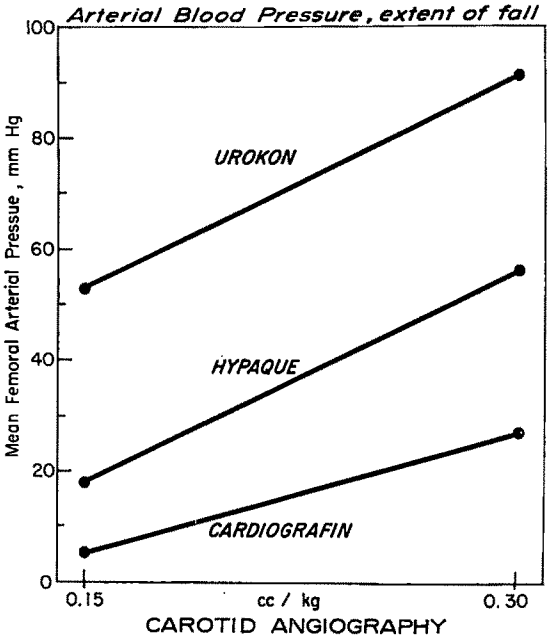


FIG. 3. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to extent of fall of arterial blood pressure. (Each point on Figure 3 through 7 is an average of the response of 6 dogs).

tion measurements is as follows: An acute change in end-expiratory CO_2 concentration at a fixed rate of ventilation is a function of the flow of blood through the lungs. This is equal to the cardiac output. A decrease in CO_2 concentration occurring before any change in respiratory quotient could become manifest must be related to a reduction in pulmonary perfusion.[21] In these experiments the acute reduction in CO_2 concentration is interpreted as a decrease in cardiac ouput. This reduction of output in the presence of an increased venous pressure, which is a function of filling pressure, is evidence of an acute reduction in the functional state of the myocardium. This might be secondary to vagal stimulation which is known to reduce myocardial performance directly as well as to reduce output by cardiac slowing.[3] The record of a typical response is shown in Figure 2.

The decisive differences in responses are presented in Table 1 for Pattern II injections.

Summarizing the Pattern II injections, cardiografin caused the least disturbance in the measured cardiovascular functions. Hypaque was intermediate. Urokon produced the most constant and severe disturbances. Although no 2 animals responded to the same degree to a given injection, the relative responses were consistent. In 30 comparisons (of 60 injections) between hypaque and cardiografin, the disturbances caused by hypaque were clearly greater in 14 instances, equal in 13, and less in only 3. In 30 comparisons between urokon and cardiografin, the differences were even greater. Urokon caused more disturbance in 28 instances, and in only 2 instances were the responses equal. At no time did cardiografin cause more disturbance than urokon. Hypaque caused greater disturbances than urokon on only 3 occasions. In 7 comparisons, the responses to hypaque and urokon were equal, and in the other 20 comparisons urokon caused more disturbance than hypaque. The differences in responses to the three contrast media and the dose-response curves for each medium are shown

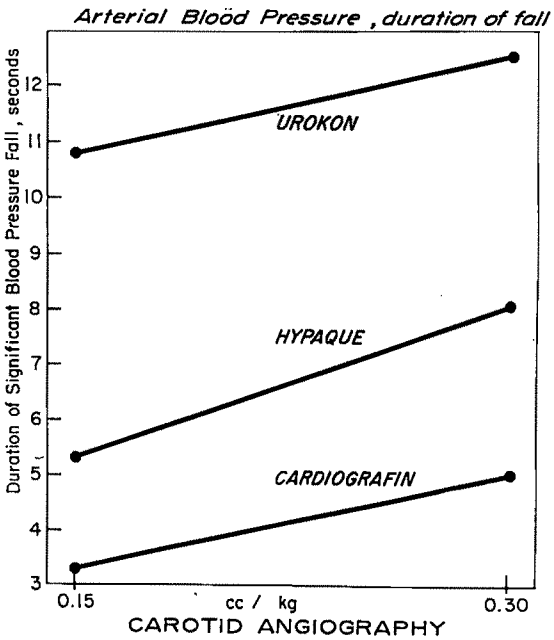


FIG. 4. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to duration of fall of arterial blood pressure.

in Figures 3 to 7. Comparison of blood pressure and heart rate for the three media at a dose of 0.15 cc./kg. are shown in 2 dogs. The responses of animal No. 289 in Figure 8 were greater than those of dog No. 290 in Figure 9. The relative responses to the three media were the same.

Of the first group of experiments (Pattern 1 injections), the types of responses were the same. The number of dogs injected and the variation of response of individual dogs do not allow more precise tabulation of data, but it was clearly seen that cardiografin was consistently least disturbing to heart rate and rhythm, the electrocardiogram, and arterial and venous blood pressure. Urokon was most consistently and severely disturbing. No overlap in the degree of response was noted between urokon and cardiografin. The responses to diodrast, hypaque and miokon were more or less of the same degree and were intermediate in magnitude between cardiografin and urokon. The responses of the three could not be distinguished consistently from each other. With these same three compounds, some overlap in response with urokon was noted at one end of the scale. At the other, less reactile end of the scale, some but less overlap in degree of response with cardiografin was observed.

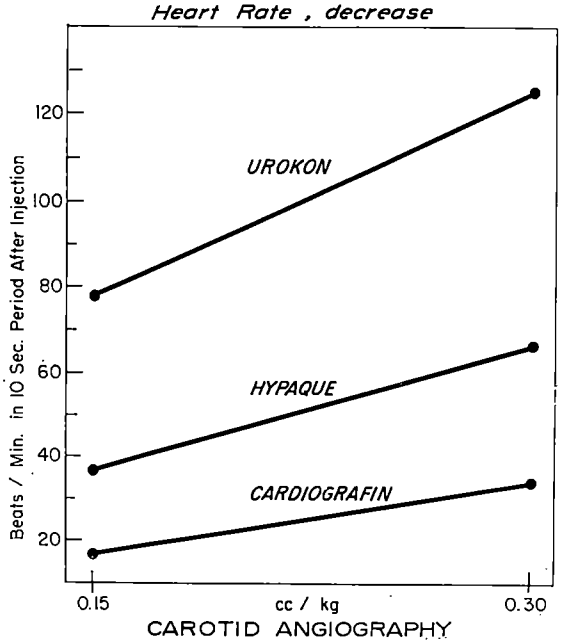


Fig. 5. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to decrease in heart rate.

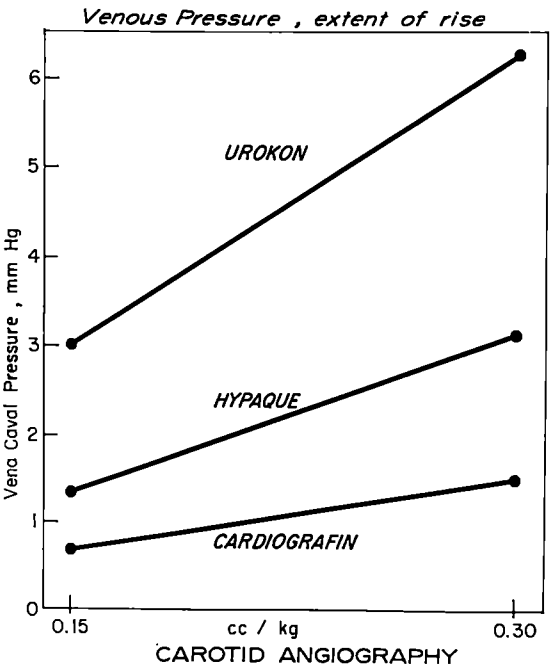


Fig. 6. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to the extent of rise of venous pressure.

COMMENT

The value of these experiments is based upon two points. First, cerebral angiographic contrast materials vary widely with respect to their pharmacologic side effects. Secondly, the observed disturbances in blood pressure, heart rate and rhythm, and cardiac output could be of clinical significance.

Toxicity of a cerebral angiographic agent is measured by disturbance in a body function or alteration of a body structure during or following its injection. Slowing of the heart and disturbance of its rhythm, lowering of arterial pressure, raising of venous pressure, and decreasing of cardiac output result from an injection of a fraction of the dose required to produce electoencephalographic abnormalities, increase in blood-brain barrier permeability or damage to the

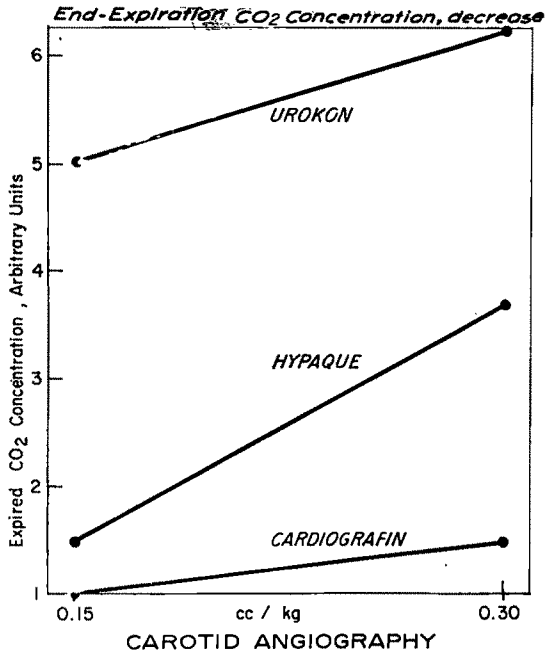


FIG. 7. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to decrease of end-expiration CO_2 concentration.

cord by experimental lumbar aortography. The amounts in the present study were 0.14 to 0.42 cc. per kg. of body weight, injected as single doses. A study of toxicity based upon change in blood-brain-barrier permeability utilized single doses of 0.7 to 3.7 cc./kg., an amount estimated to be 10 times the single injection dose in man.[27] Another blood-brain-barrier study was based upon injection into the rabbits' carotid artery of amounts 375 times as great as would be used for man.[28] Electroencephalography is not a very sensitive measure of toxicity. At least, 1.3 cc./kg. of 50 per cent diodrast was required to produce electroencephalographic abnormalities in rabbits.[2] Higher doses were more consistent in producing changes. Ingvar and Söderberg[12] found no changes in the electroencephalogram with doses comparable to those used clinically.

Experimental lumbar aortography, which has been useful in comparing toxicity of angiographic contrast agents, was based essentially on doses of 0.5 to 3.0 cc./kg. of 70 per cent concentration.[25] In clinical cerebral angiography a single injection in an adult human varies from 0.06 to 0.2 cc. per kg. of body weight, depending upon the selection of a 6 or 10 cc. injection and the body weight of the patient. Although the gross dose for the dog in the present experiments is greater than for man, consideration of the dog's carotid circulation minimizes the difference. In the dog, blood supply to the brain is derived from an internal carotid artery having a diameter a fraction of the size of the common or external carotid. The internal carotid receives a minor portion, the external carotid a major portion of the common carotid's blood flow. In man, the internal carotid artery receives a larger portion of the common carotid blood than does the external carotid. In the rabbit, when contrast solutions were injected into the internal carotid artery, blood-brain barrier damage was obtained with contrast solutions only one half as concentrated and of one tenth the volume as those producing damage in the unmodified subject.[23] Heart

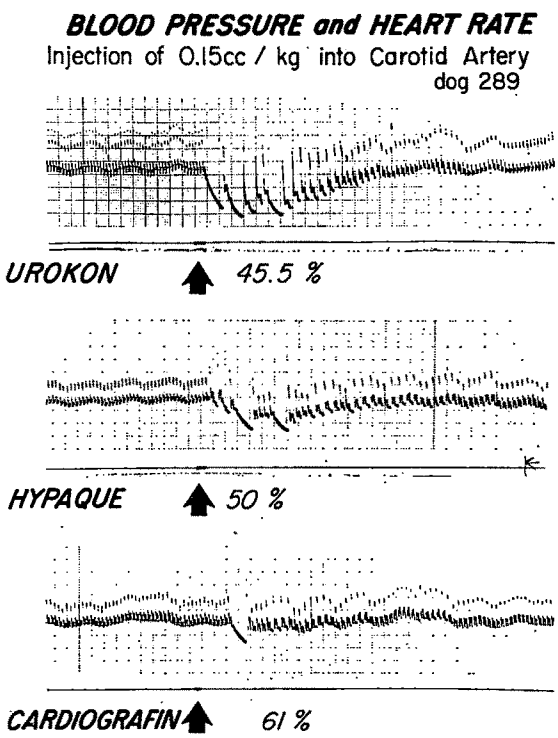


FIG. 8. Comparison of the arterial blood pressure recordings of urokon, hypaque, and cardiografin in dog 289. Equal injection times are indicated by the black bar on the time line.

rate and blood pressure changes of the same nature, as herein reported, have been produced in cats by carotid artery injections of 0.01 to 0.2 cc./kg.[16,17] To sum up, the cardiovascular responses are not only sensitive indications of toxicity, but ones based upon doses comparable to those in clinical practice.

Detailed discussion of the intricacies of the mechanism of the fall in heart rate, the arrhythmia, and the fall in blood pressure is not appropriate for this paper. The mechanism is predominantly a vagal response to brain stimulation, by the chemical nature of the contrast agent. The responses are not due to stimulation of the carotid sinus either chemically or by increase of intraluminal pressure, nor are they due to reflexes initiated by the contrast material in the external carotid system.[7,17] The responses are thought to be not dissimilar to those of vagal nature resulting from transcranial electric stimulation in man and dog.[20]

Sectioning of the vagus prevents the major acute responses. Protection from the circulatory disturbances is also available by proper adequate atropinization. Although the peripheral effects are capable of being so blocked, central stimulation is not affected. The outward early indication of trauma to the central nervous system may be concealed, but the actual irritation or damage of cerebral structures continues.

Cardiografin is the N-methylglucamine salt of 3,5 diacetamido-2,4,6 triiodobenzoic acid. Hypaque is the sodium salt of the same compound. Urokon, sodium acetrizoate, is sodium 3 monacetyl-amino-2,4, 6 triiodobenzoate. Miokon diprotrizoate is sodium 3,5 dipropionyl-amino-2,4,6 triiodobenzoate. These four compounds differ only in the nature of the side chains substituted in the 3,5 positions of the benzene ring. The remainder of the structure, the iodinated benzene ring with the iodine atoms at positions 2,4 and 6 are exactly the same. Diodrast, iodopyracet, is the sodium and diethanolamine salts of 5-diido-4 pyridone-N-acetic acid, a substantially different molecule. The chemical structures are shown in Figure 10.

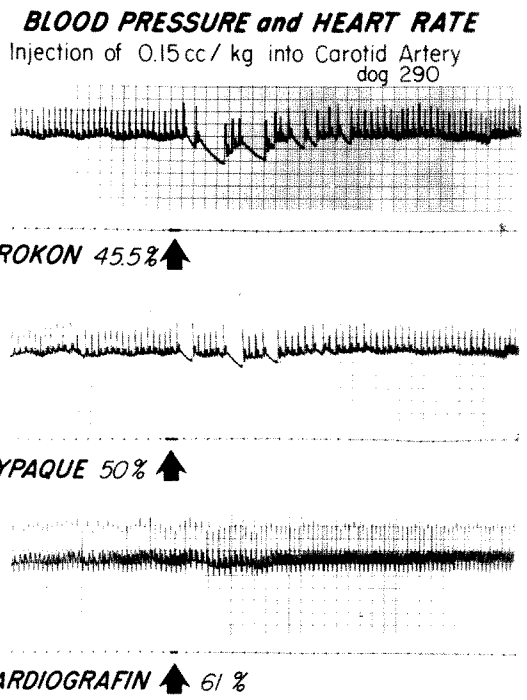


FIG. 9. Comparison similar to that in Figure 8. The disturbances are not as great, but the relative abnormality is the same.

The described disturbances in cardiovascular functions in laboratory animals may be of clinical importance during and following cerebral angiography. It is known they do occur in human cerebral angiography.[10] Unless continuous monitoring were used, they would not likely be detected, since they usually are of brief duration. For several seconds immediately after injection of the contrast material, the heart is slowed or stopped, blood pressure is low, and cardiac output is decreased. A slower passage of the contrast material through the brain could result. The contrast solution would be less subject to rapid dilution by after flowing blood. Lengthening in the time the cerebral vessels are exposed to the contrast material predisposes to other signs of toxicity, such as increase in permeability of the vessels. Lindgren, whose observations were made with a mercury manometer with its characteristic inertia, does not believe the heart and blood pressure changes occur soon enough to prolong the cerebral circulation time. Our studies, made with electronic instruments and very fast paper

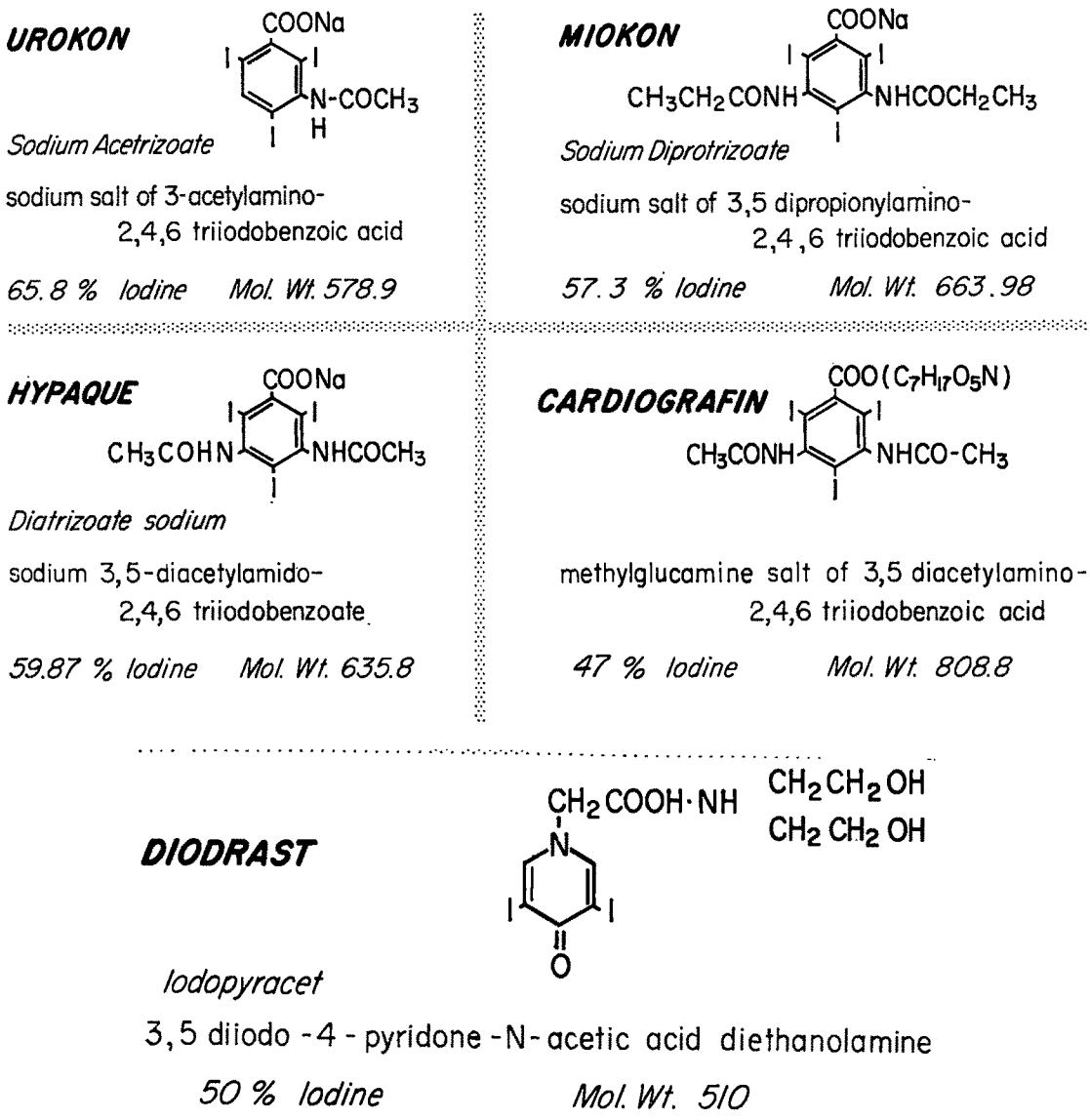


FIG. 10. Chemical structures of the 5 opaque media used in the experiments.

speeds, show the beginning of bradycardia or asystole with accompanying hypotension to occur, at times, before the completion of injection so that they could well have influence on cerebral circulation while the contrast material is on the brain. The elevation of venous pressure would also tend to slow emptying of the cerebral veins and keep contrast material in contact with vascular endothelium for a longer time.

In addition, an effect on the heart itself must be considered. A brief period of hypotension and reduced coronary perfusion may be tolerated by the healthy heart. Morbidity or mortality, however, could result in the already ischemic heart. Although it is realized that most complications of cerebral angiography are neurologic, rarely a patient in the older age group could suffer a coronary occlusion set off by a period of asystole and hypotension.

For adequate testing of contrast materials equal volumes of the same concentration must be injected. In the first experiments, 50 per cent solutions of cardiografin, diodrast, hypaque, miokon and urokon were injected each in single doses of 0.14 to 0.42 cc. per kg. of body weight. In the

later experiments, 50 per cent hypaque was selected as a standard cerebral agent. Hypaque 50 per cent, in wide clinical usage, is considered to have a relatively low incidence of complications. It is one of the newer triiodinated molecules and has good radiopacity. Primarily, radiopacity depends on the iodine content of the molecule. Equating angiographic contrast materials on the basis of their iodine content is most rational, for in this way equal radiopacity can be obtained. The expression of 50 per cent refers to the number of grams of the compound in 100 cc. of liquid, and is not to be confused with the iodine content. Iodine contents of the organic iodide contrast agents vary widely, from 47 per cent for cardiografin to urokon, iodine content 65.8 per cent. The urokon molecule, therefore, has 40 per cent more iodine than cardiografin.

For this reason, cardiografin and urokon were tested in concentrations in which their iodine contents were equated to hypaque 50 per cent (hypaque has an iodine content of 59.9 per cent, miokon 57.3 per cent, diodrast 50 per cent). Sixty-one per cent cardiografin and 45.5 per cent urokon have iodine contents equal to 50 per cent hypaque.

If the objection is made that a lesser concentration than 50 per cent will still afford satisfactory radiopacity clinically, the present studies remain valid. The experiments tested the toxicity of the different molecules on the basis of chemical concentration or on radiopacity. A 50 per cent solution was convenient as a standard because it gave measurable response, and was also in use clinically. Since some radiologists find 30 per cent urokon or 35 per cent diodrast sufficiently opaque, then cardiografin of iodine content equated to these compounds should cause even less disturbance in cardiovascular functions. Forty-two per cent cardiografin equals 30 per cent urokon in iodine content; 37 per cent cardiografin is equivalent to 35 per cent diodrast. Toxicity is no doubt reduced with lower concentrations and smaller volumes of any of the contrast agents, although we did not test solutions in the 30–35 per cent range. The important point remains, for equal radiopacity, cardiografin is the least toxic. Hypaque is intermediate, and urokon the most toxic. For cerebral angiography we advise the least toxic compound at the lowest concentration to give satisfactory opacity to the vessels. We do not believe organic iodides can be used in concentration low enough to avoid all toxic manifestations and yet offer clinically acceptable radiopacity.

Another approach to toxicity of cerebral angiographic contrast materials has been the administration of protective substances prior to injection. Glucose and procaine had been found effective in protecting the dogs' spinal cord from the damaging effects of urokon in experimental aortography.[15] When an attempt was made to use the two substances in clinical carotid arteriography, the prior intracarotid injection of procaine caused seizures in 3 of 8 patients. Administration of glucose prior to angiography did not greatly disturb the patients, but resulted in diminished roentgenographic contrast of the internal carotid system.[24] Until a more safe and reliable means of protecting against the toxic effects of a contrast material can be found, the compound with the fewest and most minimal toxic effects must be sought for and used.

Table II lists the previous experiments assessing the toxicity of contrast agents for cerebral angiography. The results closely parallel those of the present work, with few exceptions, despite a variety of methods used, and a wide dose range. Neither the present work nor the previous experiments listed in Table I can be taken as unequivocal evidence of the superiority of one contrast material over another for clinical usage. Despite pharmacologic inertness and lack of toxicity in animals, a contrast material may still give rise to complications and side effects in man. It is reasonable to assume, however, a consistent lack or paucity of toxic side effects, as based on several different criteria in animals, is a strong point in favor of a particular contrast material. We are not aware of published data

TABLE II

COMPARISON OF CEREBRAL ANGIOGRAPHIC CONTRAST MEDIA

Laboratory Method of Evaluation	Measurement of Toxicity—Degree of Derangement				
	Least ←	Intermediate			→ Most
Blood-brain-barrier permeability after carotid arteriography (Whiteleather and DeSaussure[27])	Hypaque 50%	Diodrast 35%	Urokon 30%	Urokon 50%	
Functional and anatomic changes after lumbar aortography (Tindall *et al.*[25])	Renografin 70%	Hypaque 70%	Miokon 70%	Diodrast 70%	Urokon 70%
Coronary blood flow, myocardial contractility, electrocardiogram in coronary arteriography (Guzman and West[11])	Diodrast 70%	Renografin 76%	Cardiografin 85%	Hypaque 50%	Urokon 70%
Blood-brain-barrier permeability, neurologic and respiratory changes after carotid arteriography (Winthrop-Stearns, Inc.[28])	Cardiografin 50%	Hypaque 50%	Diodrast 50%	Urokon 50%	
Cardiovascular disturbances in carotid arteriography (Lindgren *et al.*[16–18])	Hypaque 50% Miokon 50%			Triurol 50%	
Blood flow after peripheral arteriography (Lindgren and Törnell[18])	Hypaque 45%			Urokon 25–70%	
Cerebral vascular disturbances after carotid arteriography (Schmidt[22])	Perabrodil 45% Thorotrast Joduron 30% Urografin 40%			Perabrodil M 80% Joduran 70% Urografin 76%	
Blood-brain barrier permeability, histopathologic and electroencephalographic changes and seizures after carotid arteriography (Bloor *et al.*[2])	Diodrast 30–70%			Urokon 30% NeoIopax 37.5%	
Blood-brain-barrier permeability after carotid arteriography (Broman and Olsson[4–6])		Triurol 25% Umbradil 35%	Diodrast 35%		
Cerebral blood flow after carotid arteriography (Kågström *et al.*[13,14])	Hypaque 50% Miokon 50%	Umbradil 50%	Triurol 50%		

on extensive use of cardiografin in clinical cerebral angiography. The evidence from animal studies indicates the worthiness of an adequate clinical trial for this agent. Fewer complications and side effects for man are expected.

SUMMARY

Cerebral angiographic contrast media have been compared on the basis of their cardiovascular effects, after intracarotid injection in experimental animals. The abnormalities in heart rhythm, arterial and venous pressure, and cardiac output are considered a measure of the toxicity of a contrast medium. These disturbances may also be of clinical importance. Cardiografin caused the least disturbances, urokon the most, while hypaque was intermediate in effect.

Harry W. Fischer, M.D.
Department of Radiology
State University of Iowa
College of Medicine
Iowa City, Iowa

REFERENCES

1. BASSETT R. C., ROGERS, J. S., CHERRY, G. R., and GRUZHIT, C. Effect of contrast media on blood-brain-barrier. *J. Neurosurg.*, 1953, *10*, 38–47.
2. BLOOR, B. M., WRENN, F. R., JR., and MARGOLIS, G. Experimental evaluation of certain contrast media used for cerebral angiography. *J. Neurosurg.*, 1951, *8*, 585–594.
3. BROCKMAN, S. K., LINDNER, R. J., MITCHELL, J., GILMORE, J. P., and SARNOFF, S. J. Effects of vagal stimulation on ventricular function. *Fed. Proc.*, 1959, *18*, 17.
4. BROMAN, T., and OLSSON, O. Tolerance of cerebral blood vessels to contrast medium of diodrast group; experimental study of effect on blood-brain-barrier. *Acta radiol.*, 1948, *30*, 326–342.
5. BROMAN, T., and OLSSON, O. Technique for pharmaco-dynamic investigation of contrast media for cerebral angiography; effect on blood-brain-barrier in animal experiments. *Acta radiol.*, 1956, *45*, 96–100.
6. BROMAN, T., and OLSSON, O. Experimental comparison of diodonum with sodium acetrizoate with reference to possible injurious effects on blood-brain-barrier. *Acta radiol.*, 1956, *46*, 346–350.
7. FISCHER, H. W., and ECKSTEIN, J. W. Unpublished data.
8. FISHER, R. A. The Design of Experiments. Fourth edition. Oliver and Boyd, London, 1947, p. 85.
9. FOLTZ, E. L., THOMAS, L. B., and WARD, A. A., JR. Effects of intracarotid diodrast. *J. Neurosurg.*, 1952, *9*, 68–82.
10. GREITZ, T. A. Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol.*, 1956, Suppl. 140.
11. GUZMAN, S. V., and WEST, J. W. Cardiac effects of intracoronary arterial injections of various roentgenographic contrast media. *Am. Heart J.*, 1959, *58*, 597–607.
12. INGVAR, D. H., and SÖDERBERG, U. Cerebral vasomotor tone and EEG during injection of umbradil; experimental study with new method. *Acta radiol.*, 1957, *47*, 185–191.
13. KÅGSTRÖM, E., LINDGREN, P., and TÖRNELL, G. Changes in cerebral circulation during carotid angiography with sodium acetrizoate (triurol) and sodium diatrizoate (hypaque); experimental study. *Acta radiol.*, 1958, *50*, 151–159.
14. KÅGSTRÖM, E., LINDGREN, P., and TÖRNELL, G. Circulatory disturbances during cerebral angiography; experimental evaluation of certain contrast media. *Acta radiol.*, 1960, *54*, 3–16.
15. KENAN, P. D., TINDALL, G. T., MARGOLIS, G., and WOOD, R. S. Prevention of experimental contrast medium injury to nervous system. *J. Neurosurg.*, 1958, *15*, 92–95.
16. LINDGREN, P. Carotid angiography with triiodobenzoic acid derivatives; comparative experimental study of effects on systemic circulation in cats. *Acta radiol.*, 1959, *51*, 353–362.
17. LINDGREN, P., and TÖRNELL, G. Blood pressure and heart rate responses in carotid angiography with sodium acetrizoate (triurol); experimental study in cats. *Acta radiol.*, 1958, *50*, 160–174.
18. LINDGREN, P., and TÖRNELL, G. Blood circulation during and after peripheral arteriography. *Acta radiol.*, 1958, *49*, 425–440.
19. LINDNER, D. W., MARTIN, F. A., WEBSTER, J. E., and GURDJIAN, E. S. Evaluation of hypaque sodium (Win 8308-3) for cerebral angiography. *Surg. Forum*, 1956, *7*, 553–556.
20. RICHARDSON, D. J., GAHAGAN, L. H., LEWIS, W. H., JR., SHEEHAN, D., DEBEER, E. J., COLVILLE, K. I., and ELLIS, C. H. Cardiovascular responses to transcranial electric stimulation in man and dog. *J.A.M.A.*, 1959, *171*, 528–530.
21. SARNOFF, S. J., MITCHELL, J., and GILMORE, J. P. Effect of changes in atrial systole on relation between mean atrial pressure and strokework, *J. Clin. Invest.*, 1960, *39*, 1025.
22. SCHMIDT, H. W. Behaviour of pial vessels during and after intracarotid injection of roentgen contrast media. *Acta radiol.*, 1955, *44*, 100–108.
23. STEINWALL, O. Improved technic for testing effect of contrast media and other substances on blood-brain barrier. *Acta radiol.*, 1958, *49*, 281–284.
24. TINDALL, G. T., and JACKSON, J. R. Clinical evaluation of procaine and hypertonic glucose as possible adjuncts to carotid arteriography. *J. Neurosurg.*, 1960, *17*, 43–48.
25. TINDALL, G. T., KENAN, P. D., PHILLIPS, R. L., MARGOLIS, G., and GRIMSON, K. S. Evaluation of roentgen contrast agents used in cerebral arteriography. II. Application of new method. *J. Neurosurg.*, 1958, *15*, 37–44.
26. WEATHERALL, M. Pharmacological actions of some contrast media and comparison of their merits. *Brit. J. Radiol.*, 1942, *15*, 129–137.
27. WHITELEATHER, J. E., and DESAUSSURE, R. L. Experience with new contrast medium (hypaque) for cerebral angiography. *Radiology*, 1956, *67*, 537–543.
28. Winthrop-Stearns, Inc. Supplementary laboratory data on hypaque sodium.

AN IMPROVED METHOD IN DIAGNOSTIC ROENTGENOGRAPHY OF THE BREAST*

By MARVIN M. LINDELL, JR., M.D.,† *and* JAMES J. BOYLE, JR., M.D.‡
PHILADELPHIA, PENNSYLVANIA

SOFT tissue roentgenography of the female breast is neither a new nor unproved procedure. The examination, until very recently, has been utilized only by a few men. Leborgne,[13,14] Gros and Sigrist,[10] Gershon-Cohen[3–9] and others[11,12] have done much in developing the technique of breast roentgenography for diagnostic purposes. Egan,[1] in a soon to be published paper, states that he has analyzed 1,000 cases with less than a 1 per cent error. In his series the smallest demonstrable malignant lesion was 2.0 mm. in diameter. The fact that he diagnosed 19 cases of unsuspected or nonpalpable malignancies emphasizes not only the value of this examination in clinical use but suggests its merit as a survey procedure.[6,7,8]

The use of very fine grain industrial x-ray film has made possible roentgenograms of superior quality. Very small lesions are demonstrable and the characteristic calcifications within malignancies are seen in a much greater percentage of cases. The detail of the normal and the abnormal breast structure has been so improved that an accurate diagnosis is possible in most cases.

TECHNIQUE

At the Pennsylvania Hospital we have employed the method advocated by Egan and have attained comparable accuracy in diagnosis. Our cases are unselected and much the same as would be seen in any general hospital. We feel that breast roentgenography can be adeptly performed by any radiologist once he becomes familiar with the various normal and abnormal appearances. The period of self-training is not excessive; however, he should have a good knowledge of the gross anatomy and be able to produce roentgenograms of good quality.

Both breasts are roentgenographed in the superior-inferior and tangential planes. In this manner areas of abnormality are accurately localized. Type M Eastman industrial x-ray film is used in cardboard holders. The physical factors are quite variable, but range from 28 kv. and 400 mas. for the small, atrophic breast to 38 kv. and 700 mas. for very large and secretory or fibrous structures. The primary beam is unfiltered.

All patients are examined by a radiologist who records the physical findings and pertinent history, but the roentgenograms are first reviewed by another radiologist who has not seen the patient. In this manner the provisional interpretation is not influenced by any preconceived opinion. The final report is made when both radiologists, after a discussion of the roentgenographic and physical findings, concur in the diagnosis.

INDICATIONS

Breast roentgenography is not offered as a substitute for breast biopsy. It is of definite value, however, and indicated for: (1) the examination of large, pendulous breasts where palpation is notoriously inaccurate; (2) the detection of nonpalpable masses; (3) the detection of malignant change in breasts containing areas of chronic mastopathia cystica; (4) the disclosure of malignant changes in masses thought clinically to be benign; (5) the detection of the presence of bilateral breast malignancy in cases where only a unilateral mass is palpable; and (6) as a baseline record in cases of known mastopathia cystica. In these

* From the Department of Radiology, Pennsylvania Hospital, Philadelphia, Pennsylvania.
† Associate Director, Department of Radiology.
‡ Now Assistant Professor of Radiology, Hahnemann Medical College.

women periodic breast roentgenography is of extreme value because malignant lesions can long escape detection by palpation.

MATERIAL

At the Pennsylvania Hospital, breast roentgenography has been performed in a total of 206 cases. There have been 34 cases of carcinoma diagnosed by this method of which 32 have been proven correct. In 2 cases breast biopsies were not obtained and the histologic diagnosis is unknown. The first of these women had diffuse metastatic disease and the breast roentgenograms showed a typical carcinoma, but it was deep in an extremely large and fatty structure and not palpable. Because of this the surgeon did not wish to undertake a biopsy procedure. The ovaries, however, were available for histologic examination and were found to be almost completely replaced by tumor compatible in appearance with breast carcinoma.

The second woman had a palpable mass, edema and inflammation of the skin of the breast of two weeks' duration. The breast roentgenograms revealed marked increase in the density of the involved breast, indicative of edema of the breast tissue and skin. No mass was visualized. The proposed differential diagnosis was acute mastitis or inflammatory carcinoma. The mass and edema regressed within one week and only heavy cords of tissue were then palpable. This patient died recently of a cerebrovascular accident, but permission for autopsy or biopsy was not granted. It is felt that this woman had acute mastitis which regressed completely inasmuch as there was no palpable tumor or evidence of tumor metastases at the time of death.

One case of breast carcinoma escaped detection roentgenographically. There was considerable mastopathia cystica and later a carcinoma less than 2.0 mm. in diameter was found in an area most marked by fibrosis. It was detected only after multiple permanent tissue slides were made. A review of the breast roentgenograms failed to show any changes indicative of this malignancy. It was not suspected by the surgeon at the time of the operation.

In another case there was a 1.0 by 1.5 cm. lesion that was considered suspicious, but not diagnostic, of carcinoma. Biopsy revealed this to be fibrous tissue. In view of our present experience we would no longer suspect such a lesion of being malignant.

There have been 4 patients with nonpalpable breast malignancies in whom the

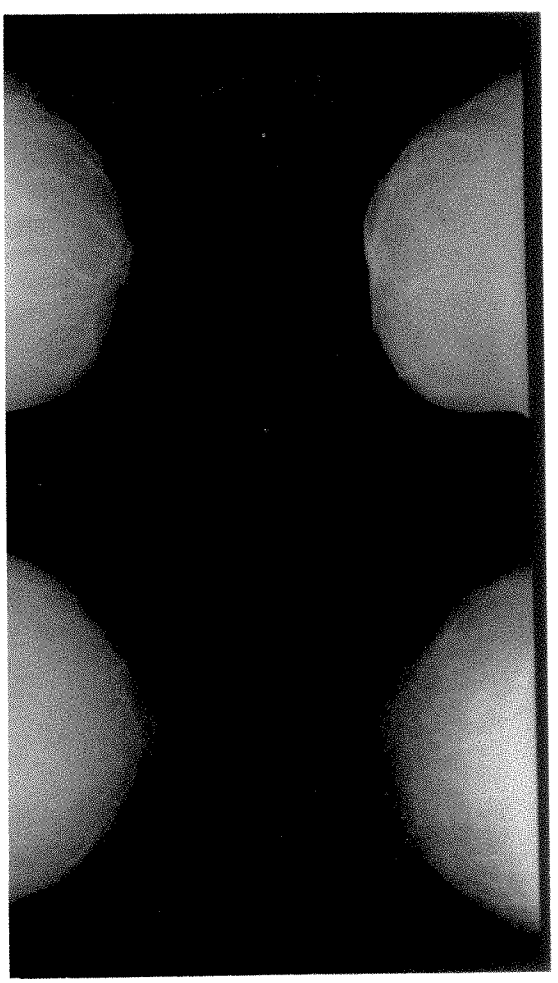


FIG. 1. Case 1. Normal postmenopausal breasts. *Top:* lateral tangential view. *Bottom:* superior-inferior view.

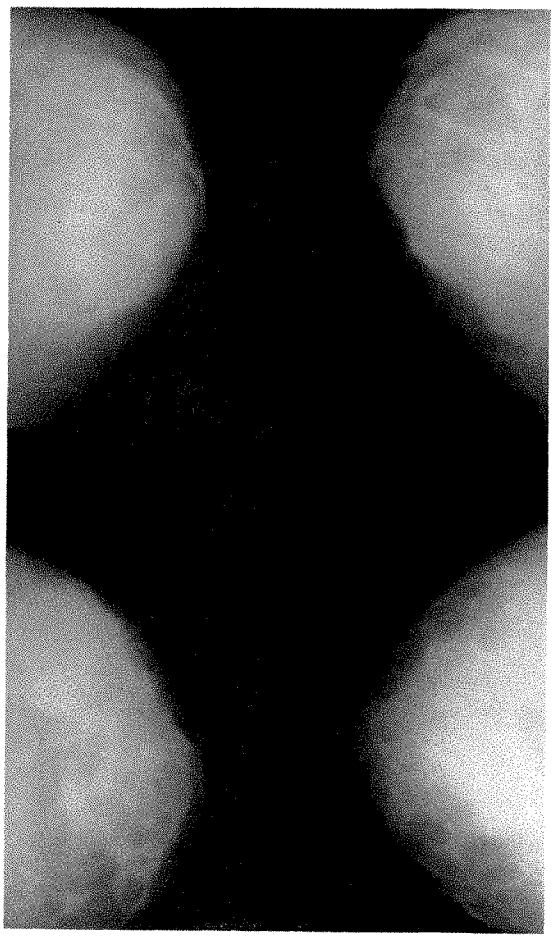


FIG. 2. Case II. Large cystic masses are visualized in both breasts. *Top:* superior-inferior view. *Bottom:* tangential view.

lesions were correctly diagnosed by breast roentgenography.

Of the benign lesions, one woman exhibited bleeding from the nipple but no palpable mass. Roentgenograms showed some minimal increased density in the subareolar tissues, but nothing indicative of malignancy. On the basis of the essentially negative roentgen appearance and the history of bleeding, a diagnosis of intraductal papillomatosis was made, which proved to be correct.

Another woman exhibited a swollen and tender breast. Roentgenograms showed edema of the breast tissues and skin, but no mass. In view of the previous case in which similar findings were present, the correct diagnosis of acute mastitis was made.

In still another woman in whom there had been direct trauma to the breast three years previously, a large cystic mass was visualized. It had only benign features and was thought to be a hematoma. Biopsy was refused.

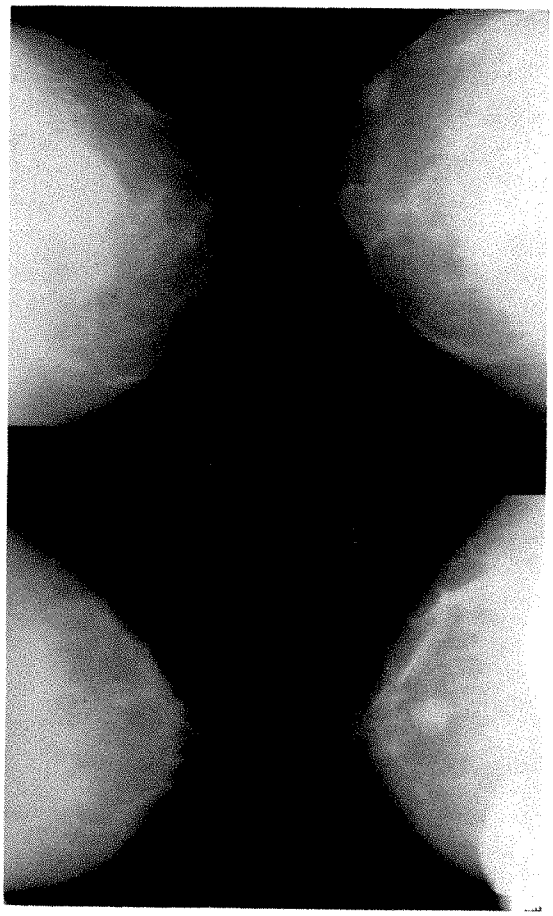


FIG. 3. Case III. Bilateral fibroadenomata. Smaller tumor on the left was not palpable.

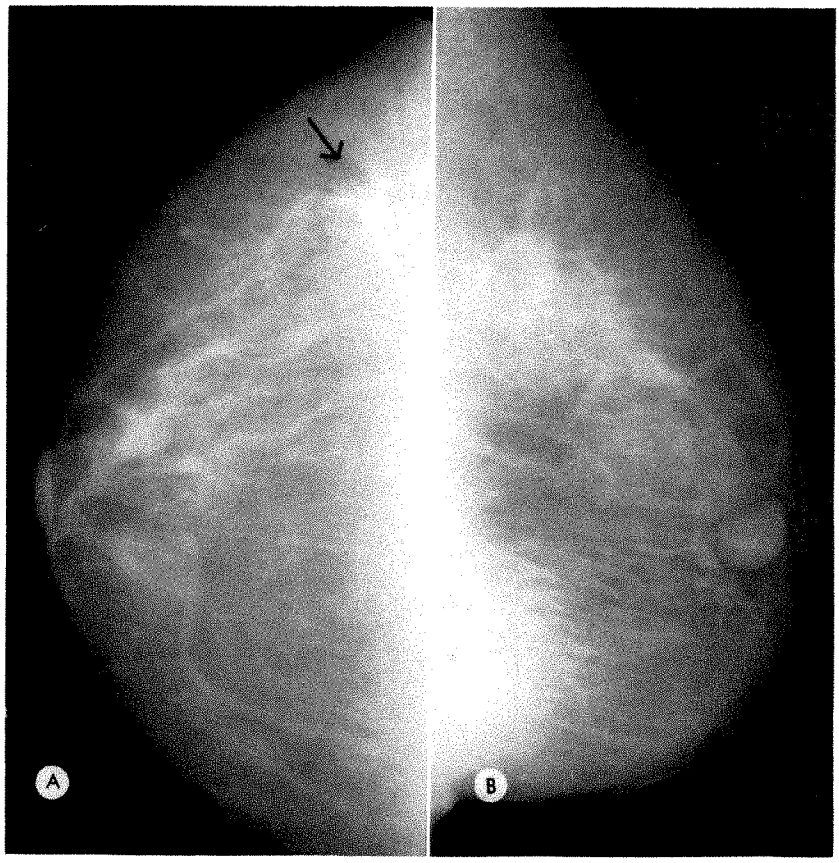


Fig. 4. Case IV. (*A*) Dense, irregular mass (arrow) typical of carcinoma. Superior-inferior view. (*B*) Note the carcinoma with radiating spicules of tumor tissue and increased vascularity. Tangential view.

ERRORS IN DIAGNOSIS

The known errors in the roentgenographic diagnosis of breast lesions in our series include one of commission and one of omission. In a third case we were unable to distinguish between acute mastitis and inflammatory carcinoma.

DIFFICULTIES IN DIAGNOSIS

The roentgenograms of secretory breasts of premenopausal women are quite difficult to interpret.[5] The tissues are very dense and irregular. Sclerosing adenosis with areas of punctate calcifications is impossible to distinguish from carcinoma.[11] Acute mastitis and inflammatory carcinoma have the same roentgen appearance and can be differentiated only when a mass with malignant characteristics is demonstrated.

Specific diagnosis is easier and more accurate in the cases of atrophic or fatty breasts of postmenopausal women. Mastopathia cystica and fibroadenomata should pose no difficulty in diagnosis.[2,3]

All cases in our series have not been proved by histologic examination since both clinically and roentgenographically many were felt to be nonsurgical conditions; however, those cases which were clinically suspected of being malignant have been biopsied and correct diagnoses have been made.

ILLUSTRATIVE CASES

Case I. This forty-four year old woman complained of pain in the left breast. She had an artificially induced menopause several years previously. The roentgenograms of the breasts (Fig. 1) demonstrated normal postmenopausal structures. There was a rather pronounced radiolucency due to fatty tissue. The normal

septa and blood vessels were well visualized and no masses or calcifications were noted.

Case II. This forty-six year old premenopausal woman had had mastopathia cystica for many years. At the time of roentgen examination she presented bilateral cystic masses which were easily palpable. These were quite painful. Roentgenograms demonstrated severe mastopathia cystica (Fig. 2). Large, dense cysts were present bilaterally as well as clusters of fibrous tissue. The cysts were fluid-filled and their borders were sharp, even though they were lobulated in contour.

Case III. This thirty-one year old premenopausal woman presented herself because of a mass in the right breast. Roentgenograms (Fig. 3) showed very dense, sharply demarcated masses typical of fibroadenomata.[2] The smaller mass on the left was not recognized by the surgeon at the time of manual examination.

Case IV. This sixty-six year old woman palpated a hard mass in her breast. The examining physician felt that, clinically, it was malignant. Roentgenograms of the involved breast (Fig. 4, *A* and *B*) showed a very dense, irregular mass in the upper outer quadrant. Spicules of tumor tissue radiated into the surrounding breast substance and there was marked increase in the number and size of the blood vessels in and about the tumor. The appearance was typical of carcinoma.

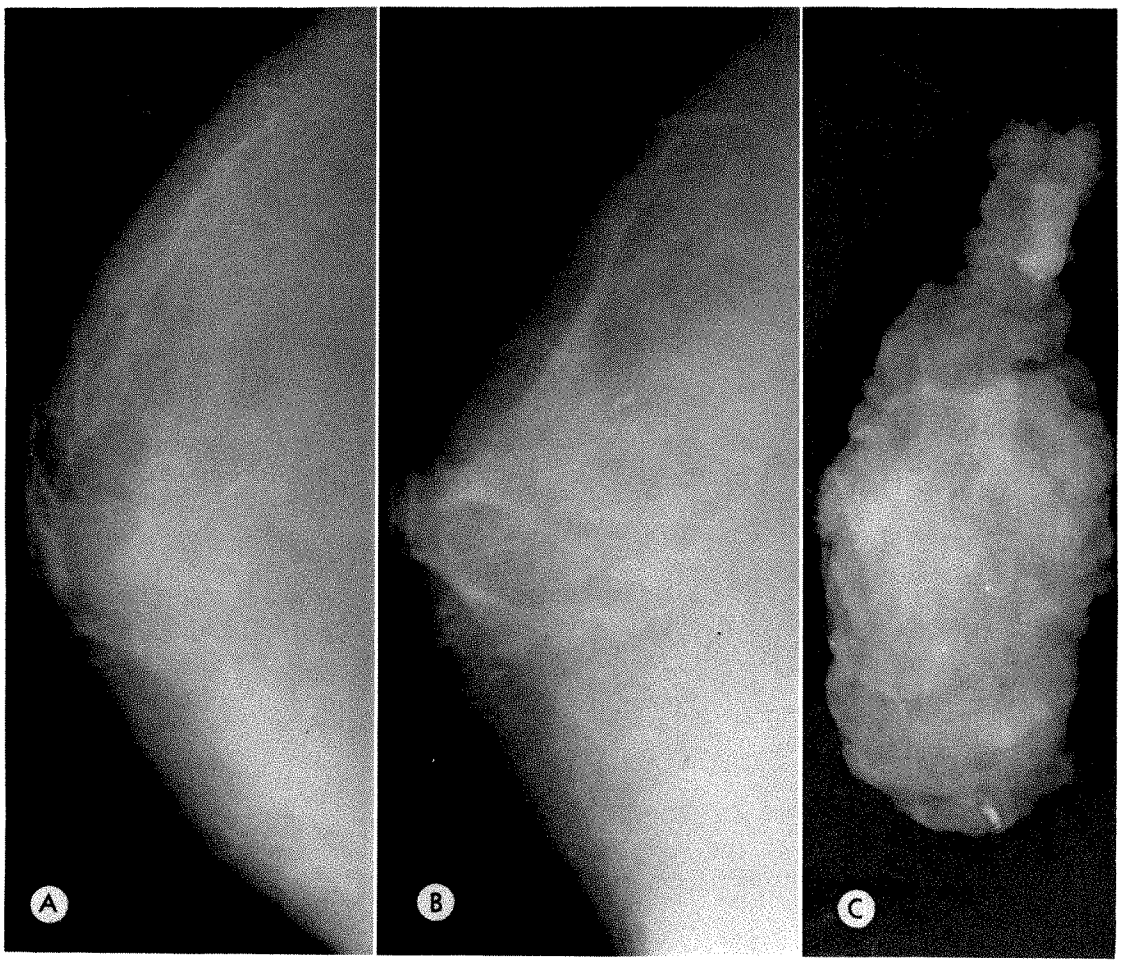


Fig. 5. Case V. (*A*) Centrally located, irregular area of calcification. Superior-inferior view. (*B*) Large calcification and smaller areas of punctate calcifications are demonstrated. The malignant mass lies deep to the calcifications. (*C*) Resected breast mass showing large area of calcification (bottom) and many smaller calcifications.

CASE V. This fifty-eight year old postmenopausal woman had an easily palpable 2.0 cm. lymph node in the right axilla. It was smooth, moderately firm and unattached to either the superficial or deep structures. It was neither painful nor tender. Repeated examinations of both breasts over a period of two months failed to reveal any palpable tumor. Roentgenograms (Fig. 5, *A* and *B*) demonstrated a large area of irregular calcification and several small punctate calcifications.[4] An ill-defined, extremely irregular mass was visualized deep to these calcifications. This type of calcification is indicative of malignancy.[4,13] Roentgenograms of the axillary lymph node showed it to be very dense and smooth. This, too, indicated malignant tissue content. The resected tumor mass (Fig. 5*C*) revealed the large area of calcification as well as many smaller calcifications. Subsequent histologic examination proved the calcifications to be areas of necrotic tumor. The roentgenographically dense axillary lymph node was the only one containing metastatic tumor.

SUMMARY

Diagnostic breast roentgenography is a valuable and accurate procedure which can be performed by utilizing roentgenographic equipment available in most radiologic departments or offices. The use of very fine grain industrial x-ray film ensures roentgenograms of superior diagnostic quality. The accuracy of this method is well documented in both the series of Egan[1] and that of the authors. Out of 206 cases examined at the Pennsylvania Hospital, 34 were diagnosed as carcinoma; of these 32 have been proven to be correct. The roentgenograms of 5 patients are presented.

Marvin M. Lindell, Jr., M.D.
Eighth and Spruce Streets
Philadelphia 7, Pennsylvania

REFERENCES

1. EGAN, R.: Personal communication.
2. GERSHON-COHEN, J., and INGLEBY, H. Roentgenography of fibroadenoma of breast. *Radiology*, 1952, *59*, 77–87.
3. GERSHON-COHEN, J., and INGLEBY, H. Roentgenography of cysts of breast. *Surg., Gynec. & Obst.*, 1953, *97*, 483–489.
4. GERSHON-COHEN, J., INGLEBY, H., and HERMEL, M. B. Roentgenographic diagnosis of calcification in carcinoma of breast. *J.A.M.A.*, 1953, *152*, 676–677.
5. GERSHON-COHEN, J., INGLEBY, H., and HERMEL, M. B. Calcification in secretory disease of breast. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *76*, 132–135.
6. GERSHON-COHEN, J., and INGLEBY, H. Roentgenography of unsuspected carcinoma of breast. *J.A.M.A.*, 1958, *166*, 869–873.
7. GERSHON-COHEN, J., INGLEBY, H., and MOORE, L. Analysis of 2,514 examinations during early phases of x-ray survey of breast. *Surg., Gynec. & Obst.*, 1958, *106*, 478–480.
8. GERSHON-COHEN, J., and INGLEBY, H. Roentgen survey of asymptomatic breasts. *Surgery*, 1958, *43*, 408–414.
9. GERSHON-COHEN, J., INGLEBY, H., and MOORE, L. Roentgenographic study of growth rate of 6 early cancers of breast. *Cancer*, 1958, *11*, 726–730.
10. GROS, C. M., and SIGRIST, R. Chapter XXII. Radiography of breast. In: Gynecologic Radiography. Edited by J. Dalsace and J. Garcia-Calderon. Paul B. Hoeber, Inc., New York, 1959.
11. INGLEBY, H., and GERSHON-COHEN, J. Adenosis of female breast. *Surg., Gynec. & Obst.*, 1954, *99*, 199–206.
12. KREMENS, V. Roentgenography of breast. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 1005–1013.
13. LEBORGNE, R. A. Diagnosis of tumors of breast by simple roentgenography: Calcifications in carcinoma. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 1–11.
14. LEBORGNE, R. A. Breast in Roentgen Diagnosis. Imp. Uruguaya, S. A., Uruguay, and Constable, London, 1953.

A DEVICE TO FACILITATE THE INTRODUCTION OF A CATHETER INTO THE TRACHEA FOR PASSIVE BRONCHOGRAPHY*

By BERNARD S. EPSTEIN, M.D.
NEW HYDE PARK, NEW YORK

ONE of the chief handicaps in performing passive bronchography is difficulty in passing the catheter into the trachea. Usually this is accomplished by inserting the catheter through the nose after the hypopharynx has been anesthetized by topical anesthesia. All too often the catheter is expelled from the trachea because of coughing, and the tip is deflected posteriorly into the esophagus. It appeared desirable, therefore, to devise an instrument through which passage of the catheter into the trachea could be performed easily under fluoroscopic control, and which

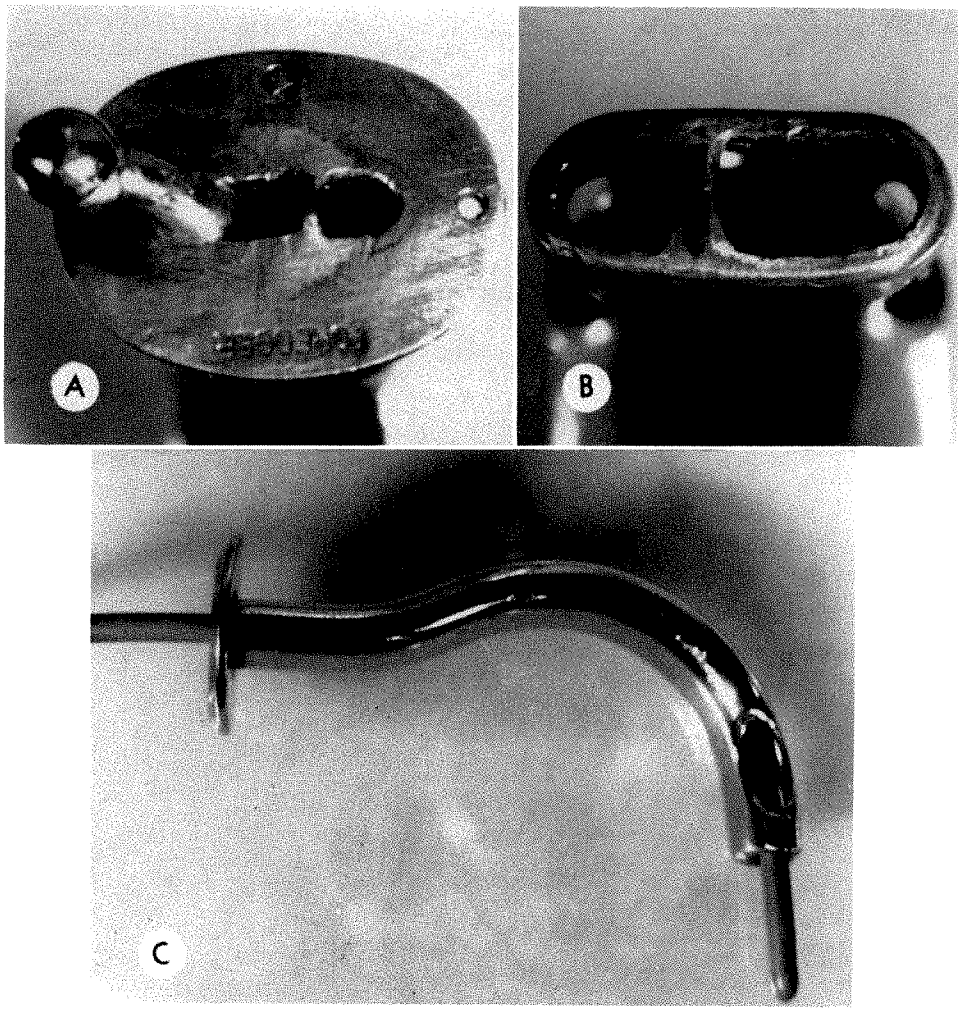


FIG. 1. (*A*) Proximal end of adapted airway. (*B*) Distal end of adapted airway. (*C*) Catheter threaded through airway.

*From the Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York.

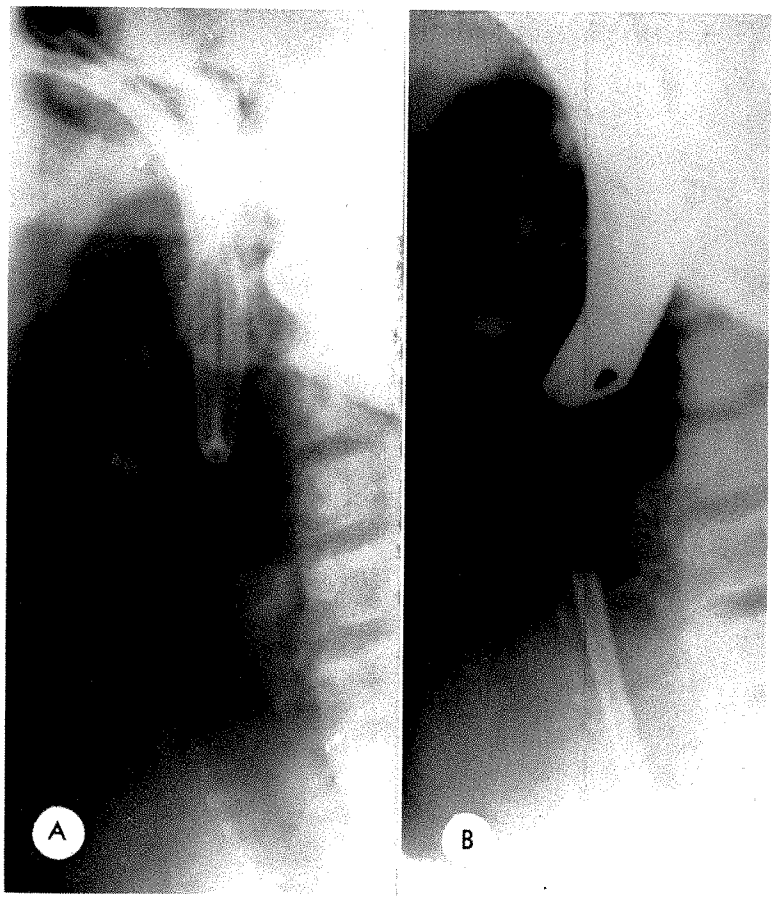


Fig. 2. (*A*) Spot roentgenogram showing catheter introduced through the nose. The tip of the catheter has turned back upon itself and is in the patient's mouth. (*B*) Catheter introduced through adaptor airway. The entrance into the trachea was rapid, and even though the patient coughed violently, the catheter was not displaced from the trachea.

would help keep it there as long as indicated.

To accomplish this, a Foregger No. 5 airway, in which a suction tube is incorporated, was adapted by welding two small bars at its proximal and distal ends, thereby establishing a guiding passageway through which a No. 14 French urethral catheter could be passed. The device is placed in the patient's mouth after local anesthesia has been applied, and under fluoroscopic control, its tip is directed towards the pharyngotracheal junction. The tip of the catheter is placed directly over the larynx, the patient is asked to breathe deeply, and the catheter is then introduced into the trachea. When this has been accomplished, a small quantity of pontocaine or cocaine is introduced into the catheter to anesthetize the carina. The study can then proceed without fear of the patient coughing back the catheter, with the confidence of having an adequate airway in place through which the catheter can be positioned as desired, and to which suction can be applied if necessary.

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Published by: CHARLES C THOMAS, PUBLISHER, 301–327 East Lawrence Avenue, Springfield, Illinois.

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* *Ex officio.*

EDITORIALS

THE SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

"WHAT a beautiful view! . . ." exclaimed awed Astronaut Alan B. Shepard, Jr., in his burst of enthusiasm upon taking his first periscopic glimpse of the land underneath the rising, arcing space ship "Freedom 7." Later, he said: "I was able to see very clearly, of course, The Florida Peninsula, Lake Okeechobee and identify Andros Island (one of the Bahamas) . . . ."

It is a fortunate coincidence that radiologists, who always have been in the vanguard of pioneering and unceasingly fostering protection against the hazards of man-made and space-made radiations, should meet on this "beautiful" peninsula, in the year of such an epoch-making event. The Sixty-second Annual Meeting of the American Roentgen Ray Society will be held, September 26-September 29, 1961, at the Deauville Hotel, in Miami Beach, the pearl of Florida.

In 1895, Röntgen made his famous discovery of the x-ray, inaugurating the era of man-made radiations. In 1896, Bequerel presented to the Paris Academy of Sciences his observations on the radioactivity of uranium compounds, initiating the era of study of the earthborn radiations, which now is rapidly expanding to the spaceborn radiations. In the spring of 1896, Henry M. Flagler, the entrepreneur, drove his puffing train through the subtropical wilderness into a tiny fishing village, laying the foundation of Miami. During the intervening six and one-half decades progress has been swift, tremendous, fabulous: swift in the application of the man-made radiations; tremendous in the understanding of earthborn and spaceborn radiations; fabulous in the full exploitation of human free enterprising ingenuity.

The isolated Miami of 1896 has literally gone "from rags to riches" by 1961, and Miami Beach has become the greatest resort city of the nation with a fantastic skyline of hotels, resplendent in ocean beaches, sands, swimming pools, magnificent gardens, and innumerable tourist attractions of the most fascinating sort. The Deauville Hotel—on the Ocean at 67th Street—in the Center of this skyline, is unique in beauty of lay-out, architecture and decor; in its modern facilities for efficiently running the Annual Meeting; and in its irresistible inducements to resort-type relaxation, pleasure and entertainment.

The forthcoming meeting will be the first since the Society was founded in 1900 to be held in Florida and Miami Beach beckons as an eminently suitable locale. The Committee on Local Arrangements under the able Chairmanship of Dr. Gerard Raap spared no time or effort in planning and organizing the many local details in the truest spirit of southern hospitality.

A preliminary program of the meeting appears elsewhere in this issue of the JOURNAL. The registration desk will be open in the Upper Convention Lobby from 2:00 P.M. to 5:00 P.M. Sunday, September 24 and thereafter daily from 8:00 A.M. to 5:00 P.M. It will close at 1:30 P.M. on Friday, September 29.

The Annual Golf Tournament for members and guests will be held at the La Gorce Country Club, Monday, September 25. The Willis F. Manges Trophy, for Eighteen Hole Medal Handicap, and the Exhibitors' Trophy, for Eighteen Hole Gross Score, and

other prizes will be awarded. The Annual Golf Dinner will follow at the La Gorce Country Club at 7:30 P.M. Arrangements have been made by the Local Golf Committee, under the Chairmanship of Dr. Virgil H. Pieck, for bus transportation to and from the Country Club, and for luncheon, golf and dinner.

The Annual Meeting of the Society will be called to order by President Harold G. Reineke at 8:30 A.M. Tuesday, September 26, in Napoleon Room No. 3. The Address of Welcome will be given by Dr. W. L. Fitzgerald, President of the Dade County Medical Association and Clinical Professor of Urology of the University of Miami, Florida. President Reineke then will install, with the assistance of Dr. Ralph M. Caulk, Chairman of the Executive Council, President-Elect Traian Leucutia as the new President of the Society. Following the Inaugural Address of President Leucutia, the Scientific Sessions will begin.

As in former years, these sessions will be held in the morning so as to afford ample time for attendance of the Instruction Courses in the mid-afternoon and for visits to the Technical Exhibits until 5:00 P.M. and the Scientific Exhibits until later in the evening.

The Program Committee has selected 48 papers to be presented at the Scientific Sessions. An impressive number of these papers encompasses topics dealing with various special procedures, bringing into sharp focus the modern trend in diagnostic roentgenology. As an appropriate expression of this trend, Dr. Harold O. Peterson, who on Tuesday evening at 8:15 will deliver in the Casanova Room this year's Caldwell Lecture, a notable event at all Annual Meetings, has chosen as his subject "The Radiologist and the Special Procedures." Dr. Peterson who is well known to members of the Society as the untiring Director of the Instruction Courses is Chairman of the Department of Radiology of the University Hospitals and Professor of Radiology of the University of Minnesota, Minneapolis, Minnesota. No one speaks with more authority on the chosen subject than does Dr. Peterson and his Lecture undoubtedly will create the greatest interest. Dr. Peterson will be introduced by Dr. J. Paul Medelman, Director of the Department of Radiology, St. Joseph's Hospital, St. Paul, Minnesota, and Clinical Associate Professor of Radiology of the University of Minnesota, a distinguished member of the Society of many attainments. The Caldwell Medal will be presented to Dr. Peterson at the conclusion of his Lecture.

Emphasizing the trend toward special procedures, valuable papers will also be presented illustrating the influence of some isotope techniques on diagnostic roentgenology.

The Friday morning Scientific Session will be devoted entirely to therapeutic problems, reflecting the more recent achievements in physics and biologic and clinical progress as well as calling attention to the hazards which may be encountered.

The Director of Instruction Courses, Dr. Harold O. Peterson, assisted by Dr. Donn G. Mosser, Associate Director, has, as usual, assembled a very representative faculty to give these courses, several of which will deal with special procedures. The detailed curriculum of the courses and a list of the members of the Faculty will be published in the August issue of the JOURNAL.

The Committee on Scientific Exhibits, of which Dr. Charles O. Heilman is the Chairman, reports that the increasing interest of the past few years continues and that the Scientific Exhibits are expected to be most instructive and of the highest quality. Several of them accompany or supplement the essays on special procedures.

The number of Technical Exhibits promises to be the largest in the Society's history, fully utilizing the available facilities, despite the fact that Dr. James C. Cook, Manager of the Annual Meeting and Mr. Clifford L. Sherrat, Honorary Member, provided additional space over that of last year's record breaking meeting in Atlantic City. In an era of great technologic prog-

ress, the Technical Exhibits represent an indispensable contribution to the success of the Annual Meeting and to enable visual documentation of the latest accomplishments, they are placed in the immediate vicinity of the Scientific Sessions.

A famous resort city, such as Miami Beach, affords unusual opportunities for social activities of the most delightful variety. This is particularly evident at this year's meeting of the American Roentgen Ray Society. The Annual Banquet which will be held Thursday evening will feature a "Nite in Caribbean." At 6:30 P.M. there will be a Cocktail Hour in the Pool Area with Music by Count Ivan and his Calypsonians. At 7:30 P.M. the banquet will start in the Casanova Room. There will be dinner dancing to the tunes of the Murray Carmen Orchestra, followed by Presentation of the 1961 Scientific Awards and an exclusive "Islander" Floor Show.

The Ladies' Committee on Local Arrangements under the able and enthusiastic Chairmanship of Mrs. Gerard Raap and Mrs. J. S. Stewart has arranged a splendid program for the ladies. On Monday, leaving the hotel at 3:30 P.M., there will be a Golf Widows' Boat Cruise to Fort Lauderdale, with Dinner at Pier 66 at 6:00 P.M., and returning by air-conditioned buses to the hotel at about 10:00 P.M. On Tuesday, starting at 10:00 A.M., there will be a Bus Tour to the Parrot Jungle, lunch at Jamaica Inn and a visit to the Seaquarium, with return to the hotel at 4:00 P.M. Wednesday will be occupied by a boat trip to Viscaya, with lunch en route, and a visit to the historic Villa Viscaya, renowned for its rare art treasures, extensive formal gardens and the many pools and fountains adorned by classic sculptures. A visit to this amazing place alone is worth a trip to the Annual Meeting. The daytime of Thursday was left open for extraprogrammatic activities and the evening was reserved for a "Nite in Caribbean."

The Officers of the American Roentgen Ray Society extend a cordial invitation to its members, all radiologists and their guests, along with their wives and families, to attend this meeting in the fabulous city of Miami Beach on the "beautiful" Florida Peninsula.

TRAIAN LEUCUTIA, M.D.
President-Elect
Harper Hospital
Detroit, Michigan



FRANKLIN B. BOGART
1894–1961

ON March 11, 1961, Dr. Franklin B. Bogart of Chattanooga, Tennessee, died at the age of sixty-six of a chronic valvular heart disease. Although he had limited his activities, he continued daily visits to his office until a few days before his death.

Franklin Bogart was born in Chattanooga, Tennessee, the son of Dr. William Moore Bogart and Keturah Thompson Bogart. He was named for his grandfather, Dr. Franklin Bogart, who practiced medicine in upper middle Tennessee. In 1916 he was graduated from the University of Wisconsin with a B.S. degree and received

his M.S. degree from the University of Wisconsin in 1917. He was graduated from Johns Hopkins University with an M.D. degree in 1919, and returned to Chattanooga in that year to establish his practice. Dr. Bogart was first associated with Dr. W. H. Cheney, who was a pioneer radiologist in this area. In 1920, Franklin Bogart married Rose Alice Harloff. He continued in the private practice of Radiology in Chattanooga until 1942, at which time he entered military service, serving as Major and later as Lieutenant Colonel in the Medical Corp AUS, as Chief of the Radiological Branch of the Lettermann General Hospital in San Francisco, Clifornia.

An ardent student of radiology and a loyal worker for organized medicine, he was a Fellow of the American College of Radiology and a Fellow of the American College of Medicine, a Diplomate of the American Board of Radiology, a member of the American Radium Society and the Radiological Society of North America, a member of the American Roentgen Ray Society and Vice-President of that organization in 1959, President of the Chattanooga and Hamilton County Medical Society and President of the Tennessee State Medical Association. He was particularly active in affairs of the Cancer Society in the State and was Co-Director of the Chattanooga Tumor Clinic, Inc., Vice-President of the Hamilton County Unit of the Tennessee Division of the American Cancer Society and served on the Board of Directors of both the local unit and the Tennessee Division for many years.

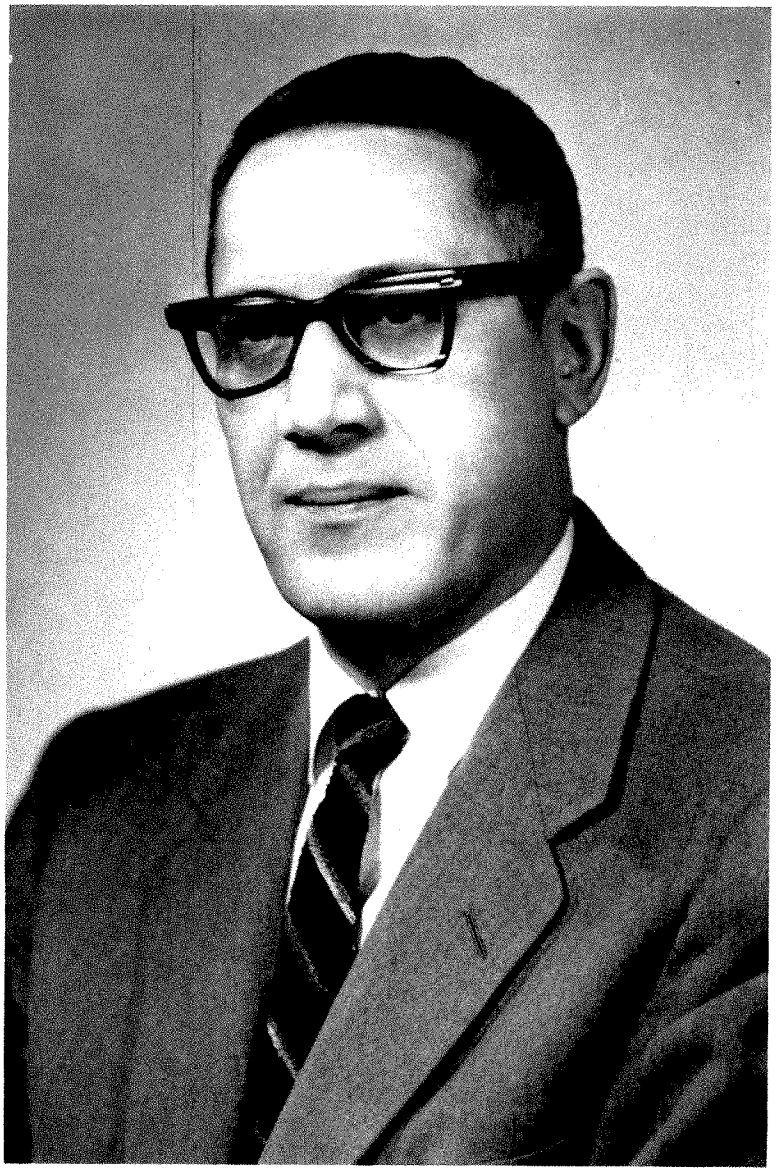
Dr. Bogart was a member of the Lookout Mountain Presbyterian Church and a member of the Fairyland Club of Lookout Mountain. He was a Rotarian and exemplified the motto, "Service above Self," in his daily practice.

Dr. Bogart was an enthusiastic gardener and in the spring the azaleas and dogwoods that thrived in his extensive gardens were a thrilling site to visitors and to passersby. In recent years, he had become interested in orchid culture and his hothouse yielded many beautiful specimens.

He is survived by his wife, Rose, and by his daughters, Alice Bogart and Mrs. David S. Smith of Westport, Connecticut, and two granddaughters.

Dr. Franklin Bogart will long be remembered as an outstanding radiologist and a loyal and honorable physician, highly respected by all of his colleagues, and will be greatly missed by them, his patients, his family and his many friends.

George K. Henshall, M.D.



DAVID M. GOULD
1914–1961

ON April 2, 1961, with the sudden death from myocardial infarction of Dr. David M. Gould, many of us lost a good friend and colleague.

Dr. Gould was born in Worcester, Massachusetts, on January 29, 1914. He attended Classical High School in Worcester and received his A.B. degree from Clark University in 1935. Upon graduation from college, he entered the Harvard Medical School where he was awarded his M.D. degree in 1939. After serving as an intern at the Boston City Hospital from 1939 to 1941, he was commissioned a Lieutenant in

the United States Public Health Service.

During the war years Dr. Gould saw service in Europe, attaining the rank of Surgeon (Major). He was assigned to duty with the United Nations Relief and Rehabilitation Administration in 1945, and in 1947 was sent to The Johns Hopkins Hospital to pursue further training in Radiology. He was appointed to the faculty of The Johns Hopkins University as an Assistant Professor in 1949, and was promoted to Associate Professor in 1950. During his years at Johns Hopkins he won the universal respect and regard of his colleagues for his keen clinical judgment and for his fine personal qualities.

In 1955 he was an exchange Professor at Guy's Hospital Medical School in London, and he lectured at Edinburgh Medical School in Scotland and at Lund University Medical School in Sweden.

In 1956, Dr. Gould was appointed Professor and Head of the Department of Radiology at the New University of Arkansas Medical Center in Little Rock. He remained there until September, 1959, at which time he came to the University of Colorado as Professor and Head of the Department of Radiology. In his three years at the University of Arkansas he developed an outstanding department which was widely recognized for its teaching, service, and research programs. At the University of Colorado, Dr. Gould again exhibited his remarkable leadership. He was particularly effective in attracting able young men to the field of Radiology, and the loyalty of all his staff, both professional and nonprofessional, testified to his leadership.

As a teacher, Dr. Gould inspired a host of students and residents by his keen judgment and modest personality. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology (Alternate Counselor to the College from Colorado, 1960) and of the American College of Chest Physicians. He was a member of the Radiological Society of North America; the American Roentgen Ray Society; the Colorado State Medical Society; the American Medical Association; the American Association of University Professors; the American Association of University Radiologists (Past-President); the American Association for the Advancement of Science; the American Society of Nuclear Medicine; the Colorado Radiological Society; and the Rocky Mountain Radiological Society.

His many friends throughout the country join with his family and his Colorado colleagues in mourning the passing of a true gentleman and scholar.

John S. Bouslog, M.D.

PRELIMINARY PROGRAM

SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Sixty-second Annual Meeting of the American Roentgen Ray Society will be held at the Deauville Hotel, Miami Beach, Florida, Tuesday through Friday, September 26 to 29, 1961.

The Executive Council will meet Sunday, September 24, 9:00 A.M. in the Normandy Room.

On Monday, September 25, the Annual Golf Tournament for Members and Guests —for the Willis F. Manges Trophy and the Exhibitors' Trophy—will be held at the La Gorce Country Club, followed by the Annual Golf Dinner at 7:30 P.M. at the same place.

The Scientific Sessions will be held in the morning from 8:30 A.M. to 12:30 P.M., Tuesday through Friday in Napoleon Room No. 3. The Caldwell Lecture will be delivered by Dr. Harold O. Peterson on Tuesday evening at 8:15 P.M. in the Casanova Room. Dr. Peterson will be introduced by Dr. J. Paul Medelman.

The Instruction Courses will be held in the afternoon from 3:00 P.M. to 4:30 P.M., Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M., Friday. The detailed plan of the Instruction Courses will be published in the August issue of the JOURNAL.

The Scientific Exhibits will be set up in the Peacock Alley and Upper Convention Lobby. They will open daily at 8:00 A.M. and will close evenings at 8:00 P.M. Tuesday, 9:00 P.M. Wednesday, and 7:30 P.M. Thursday.

The Technical Exhibits will be located in Napoleon Rooms No. 1 and 2, immediately adjacent to the meeting room of the Scientific Sessions. They will open at 8:00 A.M. and will close at 5:00 P.M., Tuesday through Thursday.

Both the Scientific Exhibits and Technical Exhibits will remain open Friday from 8:00 A.M. until 1:00 P.M.

The Annual Banquet will be held Thursday evening at 7:30 P.M. in the Casanova Room, preceded by a Cocktail Hour beginning at 6:30 P.M. in the Pool area.

The Program Committee under the Chairmanship of President-Elect Traian Leucutia has arranged the following program for the Scientific Sessions.

Tuesday, September 26, 1961
Napoleon Room No. 3

8:30 A.M. Call to Order, Sixty-second Annual Meeting: Harold G. Reineke, M.D., Cincinnati, Ohio, President. Address of Welcome: Dr. W. L. Fitzgerald, President of the Dade County Medical Association; Clinical Professor of Urology of the University of Miami, Miami, Florida.

Installation of President-Elect, Traian Leucutia, M.D., Detroit, Michigan, by Harold G. Reineke, M.D., President, Cincinnati, Ohio, and Ralph M. Caulk, M.D., Chairman of the Executive Council, Washington, D. C.

Inaugural Address: President Traian Leucutia, M.D.

9:10 A.M.

Presiding: Traian Leucutia, M.D.
Detroit, Michigan

1. The Significance of Protein Binding of Contrast Media in Roentgen Diagnosis. Elliott C. Lasser, M.D. (by invitation), Pittsburgh, Pa., Richard S. Farr, M.D. (by invitation), Pittsburgh, Pa., Gerald P. Rodnan, M.D. (by invitation), Pittsburgh, Pa., Tak Fujimagari, M.D. (by invitation), Pittsburgh, Pa., and Walter R. Tripp, M.D. (by invitation), Pittsburgh, Pa.
2. Interesting Aspects of Cerebral Angiog-

graphy with Emphasis on Some Unusual Congenital Variations. C. Richard Perryman, M.D., D.Sc. (Med.), Pittsburgh, Pa., C. H. Gray, M.D. (by invitation), Pittsburgh, Pa., and Raymond W. Brust (by invitation), Pittsburgh, Pa.

3. Angiography As an Aid in Determining Resectability of Lung Cancer. D. E. Sanders, M.D. (by invitation), Toronto, Ont., Can., N. C. Delarue, M.D. (by invitation), Toronto, Ont., Can., and George Lau, M.D. (by invitation), Toronto, Ont., Can.
4. Radiographic Aspects of Anomalous Pulmonary Venous Drainage. Melvin M. Figley, M.D., Seattle, Wash., Leon A. Phillips, M.D. (by invitation), Seattle, Wash., Robert A. Bruce, M.D. (by invitation), Seattle, Wash., Warren G. Guntheroth, M.D. (by invitation), Seattle, Wash., and K. Alvin Merendino, M.D. (by invitation), Seattle, Wash.
5. The Cinefluorographic Approach to the Diagnosis of Pericardial Effusion. Joseph Jorgens, M.D., Ph.D. (by invitation), Minneapolis, Minn., and Robert R. Kundel, M.D. (by invitation), Minneapolis, Minn.
6. The Radiographic Manifestations of Aortic Valvular Disease. E. C. Klatte, M.D., (by invitation), Indianapolis, Ind., J. A. Campbell, M.D., Indianapolis, Ind., W. E. Judson, M.D. (by invitation), Indianapolis, Ind., and P. R. Lurie, M.D. (by invitation), Indianapolis, Ind.
7. Cardiac Ventriculography (Transaortic Catheter Opacification of the Left Ventricle). J. Stauffer Lehman, M.D., Philadelphia, Pa., Joseph N. Debbas, M.D. (by invitation), Philadelphia, Pa., and James J. Boyle, Jr., M.D. (by invitation), Philadelphia, Pa.
8. Flexible Tubing for Flow-Guided Cardiovascular Catheterization and Visualization. Charles T. Dotter, M.D., Portland, Ore., and Kurt R. Straubbe, M.D. (by invitation), Portland, Ore.
9. Extra Large Field Angiography Technique and Results. Albert Jutras, M.D. (by invitation), Montreal, Que., Can., Marcel Longtin, M.D. (by invitation), Montreal, Que., Can., and Paul Roy, M.D. (by invitation), Montreal, Que., Can.
10. Physical Aspects of High Pressure Angiography. Fred C. Shipps, M.D., Portland, Ore.

Tuesday Evening, September 26, 1961
Eight-fifteen o'clock
Casanova Room

THE CALDWELL LECTURE

The Radiologist and the Special Procedures

by

Harold O. Peterson, M.D.

Chairman of Department of Radiology of the University Hospitals, and Professor of Radiology, University of Minnesota, Minneapolis, Minn.

Introduction:

J. Paul Medelman, M.D., Director of Department of Radiology, St. Joseph's Hospital, St. Paul, Minnesota, and Clinical Associate Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

Wednesday, September 27, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Clyde A. Stevenson, M.D.
Spokane, Washington

11. Hemoglobin S-C Disease. Joshua A. Becker, M.D. (by invitation), Philadelphia, Pa.
12. Radiographic and Clinical Appraisal of Sickle Cell-Hemoglobin C Disease. Jack Reynolds, M.D. (by invitation), Dallas, Tex.
13. Radiologic Manifestations of Juvenile Rheumatoid Arthritis. William Martel, M.D. (by invitation), Ann Arbor, Mich., John F. Holt, M.D., Ann Arbor, Mich., and James T. Cassidy, M.D. (by invitation), Ann Arbor, Mich.
14. Cineradiographic Studies of Patients with Cervical Spine Fusion. Malcolm D. Jones, M.D. (by invitation), San Francisco, Calif.
15. Anesthesia for Bronchography Utilizing Aerosol-Nebulizer. William Molnar, M.D. (by invitation), Columbus, Ohio, and John Prior, M.D. (by invitation), Columbus, Ohio.
16. Postoperative Pulmonary Atelctasis. J. Pollard, M.D. (by invitation), Boston, Mass., and H. Bendixen, M.D. (by invitation), Boston, Mass.

17. Breast Cancer Detection by Periodic X-Ray Examination—Five Year Survey. J. Gershon-Cohen, M.D., D.Sc. (Med.), Philadelphia, Pa., M. B. Hermel, M.D., Philadelphia, Pa., and Simon M. Berger, M.D. (by invitation), Philadelphia, Pa.
18. Lymphangiography; Its Role in Diagnosis and Therapy. Sidney Wallace, M.D. (by invitation), Philadelphia, Pa., Laird Jackson, M.D. (by invitation), Philadelphia, Pa., and Roy Greening, M.D., Philadelphia, Pa.
19. Renal Involvement in Lymphomas and Leukemias in Adults. Jerome H. Shapiro, M.D. (by invitation), New York, N. Y., G. Craig Ramsay, M.D. (by invitation), New York, N. Y., Harold G. Jacobson, M.D., New York, N. Y., and Charles Botstein, M.D. (by invitation), New York, N. Y.
20. Curable Arterial Hypertension Due to Renal Artery Lesions, Including Fibromuscular Hyperplasia. A. J. Palubinskas, M.D. (by invitation), San Francisco, Calif.
21. The Radioactive Kidney Test As a Screening Procedure in Hypertension. A. Weber, M.D. (by invitation), Boston, Mass., and T. Agard, M.S. (by invitation), Boston, Mass.
22. Coarctation of the Renal Artery with "Notching" of the Ureter: A Roentgenologic Sign of Unilateral Renal Disease As a Cause of Hypertension. Mordecai Halpern, M.D. (by invitation), New York, N. Y., and John A. Evans, M.D., New York, N. Y.
23. The Effect of (Voluntary) Bladder Distention on the Intravenous Pyelogram: An Experimental Study. J. S. Dunbar, M.D., Montreal, Que., Can., D. W. MacEwan, M.D. (by invitation), Montreal, Que., Can., and Bernedette Nogrady, M.D. (by invitation), Montreal, Que., Can.
24. The Radiation Hazard. Gerald M. McDonnel, M.D. (by invitation), Los Angeles, Calif.

Thursday, September 28, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Ralph M. Caulk, M.D.
Washington, D. C.

25. Opacification Studies of Normal and Abnormal Paranasal Sinuses. Lewis E. Etter, M.D., Pittsburgh, Pa.
26. Roentgen Demonstration of Cranial Nerve Metastases in Tumors of the Head and Neck. Gerald D. Dodd, M.D. (by invitation), Houston, Tex.
27. Intra-aural Radiography of the Fenestra Ovale. O. Arthur Stiennon, M.D. (by invitation), Madison, Wis., and James F. Land, M.D. (by invitation), Madison, Wis.
28. Skeletal Abnormalites Associated with Gonadal Dysgenesis. Nathaniel Finby, M.D., New York, N. Y., and Reginald M. Archibald, M.D. (by invitation), New York, N. Y.
29. Chondromyxoid Fibroma of Bone. Bernard Turcotte, M.D. (by invitation), Rochester, Minn., David G. Pugh, M.D., Rochester, Minn., and David C. Dahlin, M.D. (by invitation), Rochester, Minn.
30. What Is Multiple Myeloma? Robert Phelps Barden, M.D., Philadelphia, Pa.
31. Roentgen Findings in Diseases with Abnormal Calcium Deposition. Theodore F. Hilbish, M.D., Bethesda, Md., and Frederic C. Bartter, M.D. (by invitation), Bethesda, Md.
32. Radiologic Findings in the Small Bowel in Scleroderma. James Reinhardt, M.D. (by invitation), Durham, N. C., and William F. Barry, Jr., M.D., Durham, N. C.
33. Duodenal Roentgenographic Manifestations of Non-Tropical Sprue. John E. Shick, M.D. (by invitation), Detroit, Mich.
34. Fluid-Filled Loops in Intestinal Obstruction. J. L. Williams, M.D. (by invitation), Danville, Pa.
35. The Use of Silicone Foam As an Enema in Examining the Human Sigmoid Colon. Galen B. Cook, M.D. (by invitation), St. Louis, Mo., and Alexander R. Margulis, M.D., St. Louis, Mo.
36. Scintillation Scanning of the Liver. Frederick J. Bonte, M.D., Dallas, Tex., J. S. Krohmer, M.A., Dallas, Tex., Ernest A. Elmendorf, M.D. (by invitation), Dallas, Tex., Norman L. Presley, M.D. (by invitation), Dallas, Tex., and Giles J. Andrews, M.D. (by invitation), Dallas, Tex.

Friday, September 29, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Robert C. Pendergrass, M.D.
Americus, Georgia

37. Analysis of the Results of X-Ray Therapy by Region in Malignant Lymphoma. Lillian M. Fuller, M.D. (by invitation), Houston, Tex.
38. Radiation Therapy of Chronic Lymphatic Leukemia—a Statistical Analysis. James C. Cook, M.D., Detroit, Mich., and Walter Romano, M.D. (by invitation), Detroit, Mich.
39. Results in Squamous Cell Carcinoma of the Cervix: A Comparison of Orthovoltage and Cobalt 60 Radiation. George Cooper, Jr., M.D., Charlottesville, Va., and David H. Williams, M.D. (by invitation), Charlottesville, Va.
40. Evaluation of Complications Encountered in the Treatment of Malignancy with the Telecobalt Beam, with Recommendations for Measures to Avoid Them. Damon D. Blake, M.D. (by invitation), Winston-Salem, N. C., I. Meschan, M.D., Winston-Salem, N. C., and W. A. Covell, M.D. (by invitation), Winston-Salem, N. C.
41. Osteoradionecrosis of the Mandible. Bernard Roswit, M.D. (by invitation), Bronx, N. Y.
42. Dose-Time Relationships in Clinical Radiotherapy; Clinical Observations and Rabiobiological Correlation. J. Robert Andrews, M.D., Bethesda, Md., Kirkland C. Brace, M.D. (by invitation), Bethesda, Md., and Roger J. Berry, M.D. (by invitation), Bethesda, Md.
43. The Second Half-Value Layer and the Homogeneity Factor. E. Dale Trout, B.S., D.Sc., Milwaukee, Wis., John P. Kelly, B.S. (by invitation), Milwaukee, Wis., and Arthur C. Lucas, B.S. (by invitation), Milwaukee, Wis.
44. The Effect of Lowering Temperature to Give Skin Sparing and Increase Depth Dose with Conventional X-Rays (300 kv.). Edwin J. Liebner, M.D. (by invitation), Chicago, Ill., Walter Moos, Ph.D. (by invitation), Chicago, Ill., Martin Hochhauser, M.D. (by invitation), Chicago, Ill., and Roger Harvey, M.D., Chicago, Ill.
45. Clinical Experience with Roentgen Therapy under High Oxygen Tension. W. B. Seaman, M.D., New York, N. Y., C. S. Sanger, M.D. (by invitation), New York, N. Y., N. DuV. Tapley, M.D. (by invitation), New York, N. Y., H. W. Jacox, M.D., New York, N. Y., and H. L. Atkins, M.D. (by invitation), New York, N. Y.
46. Ten Years' Experience with Low Megavolt Electron Therapy Beam. M. I. Smedal, M.D., Boston, Mass., D. O. Johnston, M.D. (by invitation), Boston, Mass., F. A. Salzman, M.D., Boston, Mass., J. G. Trump, Sc.D., Cambridge, Mass., and K. A. Wright, M.S. (by invitation), Cambridge, Mass.
47. The Use of P^{32} in the Differential Diagnosis of Ocular Neoplasms. C. I. Thomas, M.D. (by invitation), Cleveland, Ohio, J. P. Storaasli, M.D., Cleveland, Ohio, and H. L. Friedell, M.D., Cleveland, Ohio.
48. Newer Trends in Radiotherapy. Justin J. Stein, M.D. (by invitation), Los Angeles, Calif.

NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

At a meeting of the American Radium Society, held at the Broadmoor Hotel in Colorado Springs, Colorado, on May 10–14, 1961, the following officers were elected: President, Robert L. Brown, Atlanta, Ga.; President-Elect, Gilbert H. Fletcher, Houston, Texas; 1st Vice-President, Ralph F. Phillips, New York, N. Y.; 2nd Vice-President, Ethlyn Trapp, Vancouver, B. C., Can.; Secretary, Charles G. Stetson, Englewood, N. J.; and Treasurer, Joseph H. Farrow, New York, N. Y.

The Forty-fourth Annual Meeting of the Society will be held at the Waldorf-Astoria Hotel in New York City on April 1–4, 1962.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

The twenty-third midsummer radiologic conference of The Rocky Mountain Radiological Society will be held at the Denver Hilton Hotel, Denver, Colorado, August 10, 11, and 12, 1961. The guest speakers will be: Harold G. Jacobson, M.D., Director, Division of Diagnostic Radiology, Montefiore Hospital, 210th Street and Bainbridge Rd., New York 67, N. Y.; Walter T. Murphy, M.D., Director of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo 3, N. Y.; and William B. Seaman, M.D., Director, Department of Diagnostic Radiology, Columbia-Presbyterian Medical Center, 622 West 168th Street, New York 32, N. Y.

Friday, the 11th, has been dedicated to Dr. Eugene Pendergrass. The Society will honor him for his vast contributions to radiology on the year of his retirement. On this day the program will consist of papers by former residents of Dr. Pendergrass. These speakers will be: Kenneth D. A. Allen, M.D., Denver, Colorado; J. Gershon-Cohen, M.D., Philadelphia, Pennsylvania; Roderick L. Tondreau, M.D., Meadowbrook, Pennsylvania; James C. Katterjohn, M.D., Indianapolis, Indiana; John F. Weigen, M.D., Palo Alto, California; Gilbert W. Heublein, M.D., Hartford, Connecticut; John M. Dennis, M.D., Baltimore, Maryland; C. Richard Perryman, M.D., Pittsburgh, Pennsylvania; Harold Atkins, M.D., New York, New York; Robert P. Barden, M.D., Philadelphia, Pennsylvania; Vern W. Ritter, M.D., Santa Rosa, California; Ellwood W. Godfrey, M.D., Princeton, New Jersey; Nathan P. Salner, M.D., Philadelphia, Pennsylvania; and J. Robert Andrews, M.D., Bethesda, Maryland.

At the annual banquet a plaque will be presented to Dr. Pendergrass in recognition of this occasion. A plaque will also be presented to Dr. Milton Berg, Bismarck, North Dakota, President of the Radiological Society of North America. This will be presented in recognition of his many years of faithful service to the Rocky Mountain Radiological Society and his many contributions to medicine and radiology.

WORLD ASSOCIATION OF ACADEMIC PROFESSORS OF MEDICAL RADIOLOGY

A meeting of the World Association of Academic Professors of Medical Radiology will be held at the Cantonal Hospital, Zürich, Switzerland, from Friday, October 13, until Sunday, October 15, 1961. A banquet will be given on Saturday, October 14, 1961, by the President of the Association, Professor H. R. Schinz, M.D.

Report of intended attendance at the meeting as well as requests for information concerning hotel facilities, etc. should be addressed to the Secretary of the Association, Prof. U. Cocchi, M.D., Kantonsspital, Zürich, no later than June 30, 1961.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

NUCLEAR EXPLOSIONS AND THEIR EFFECTS. Revised and enlarged edition. Foreword by Jawaharlal Nehru. Cloth. Price, $4.00. Pp. 340 and 5 charts. The Publications Division, Ministry of Information and Broadcasting, Post Box No. 2011, Old Secretariat, Delhi 8, India, 1958.

In the foreword to the first edition of this book, which was published in June, 1956, Nehru stated: "About a year ago I suggested to the Defence Science Organization of the Government of India that an objective study might be made with the material available of the consequences of the use of nuclear, thermonuclear and other weapons of mass destruction." D. S. Kothari of the Indian Defence Science Organization was entrusted with this task and with the assistance of H. Bhabha, in charge of the atomic energy work, and V. R. Khanolkar, head of the cancer research institute, collected the material upon which this revised and almost completely rewritten edition is based.

After a few historical remarks on the development of nuclear energy, starting with the discovery of x-rays by Röntgen in 1895, the authors describe in detail fission and fusion explosions, fission energy and radioactivity of fission products, and the effects of atomic bombs and of high-yield explosions. This is followed by detailed and valuable presentations of the subjects on long-term radiologic hazards, such as global fallouts and notably of genetic effects. A rather extensive appendix gives further information on fission explosion and critical size, thermonuclear reactions, catalyzed fusion reaction, dose rates from radioactive sources inside and outside the body, and nuclear explosions and the weather.

This book, which also has been published in Germany and Japan, gives an excellent presentation of the dangers of large nuclear bombs. It is written with great clarity in a style that the reader who knows little about physics can well understand. It can be highly recommended.

OTTO GLASSER, PH.D.

NUCLEAR REACTORS FOR POWER GENERATION. Edited by E. Openshaw Taylor, B.Sc., D.I.C., M.I.E.E., M.Am.I.E.E., F.R.S.E., Assistant Professor of Electrical Engineering, Heriot-Watt College, Edinburgh, Scotland. Cloth. Price, $7.50. Pp. 144, with 51 illustrations. Philosophical Library Inc., 15 East Fortieth Street, New York 16, N. Y., 1958.

This book is based on a series of advanced lectures delivered at the Heriot-Watt College in Edinburgh, Scotland, by the editor, E. O. Taylor, with the assistance of J. H. Bowen, W. H. J. Childs, J. M. Kay, W. L. Kidd, and A. B. McIntosh. After introductory remarks about the world energy requirements and present fuel reserves, the nuclear energy programs of America, Great Britain, Russia and some smaller European countries are discussed. This is followed by chapters on fundamental nuclear physics, basic design and various types of nuclear reactors, building materials used in reactors, safety and instrumentation and the economics of different types of reactors.

The book is well written and printed and the numerous line drawings are very helpful in interpreting the text. It should be read by all those who are interested in atomic reactors, whether they are used for the production of power or of isotopes.

OTTO GLASSER, PH.D.

LE RADIOCALCIUM DANS L'ÉTUDE DES Os. By R. Ponlot, Chercheur agréé de l'Institut Interuniversitaire des Sciences Nucléaires, Chef de Travaux à l'Université de Louvain. Paper. Pp. 254, with 86 illustrations. Editions Arscia S.A., 60, rue de l'Etuve, Bruxelles, Belgium; Masson et Cie, 120 Boulevard Saint-Germain, Paris 6e, France, 1960.

In 1955, Dr. J. Vincent, in a monograph titled "Experiments on the Constitution of the Adult Bone," had demonstrated that, in order to obtain good interpretation of a contact autoradiograph of the osseous tissue, it is necessary to compare it not only with the histologic section but also with the microradiograph of the bone. Because of this, the interest has been focussed on the mineral portion of the bone entailing the utilization of Ca^{45} isotope studies.

Ponlot has pursued such studies since 1955,

performing numerous investigations, mainly on the physiopathology of the bone; *i.e.*, the role of the osseous skeleton in the general metabolism of calcium in the system. In the present excellent monograph he expounds the results of his Ca^{45} investigations with regard to the histology of bone aiming to establish the morphologic significance of autoradiographs performed *in vivo*.

The first five chapters deal with the histologic pattern of the compact adult bone and the structural changes of the haversian system. The affinity of collagen for mineral salts, revealed by certain experiments *in vitro*, is probably not the only factor determining the calcification *in vivo*.

The next five chapters discuss autoradiographic studies of the adult compact bone made 7 hours, 12 days, and 6 weeks following injection of Ca^{45}, and an attempt is made to interpret the phenomena of calcification and mineral salt exchanges. The remaining ten chapters are devoted to detailed investigations of the bone growth particularly of the tibia and vertebra.

The author describes *in extenso* the numerous investigations performed and uses many pertinent illustrations of the finest quality to emphasize the important points of the discussion. He concludes by defining the possible applications both for experimental and clinical investigations in Orthopedic Surgery and the role which Sr^{90} and Ca^{45} respectively may occupy in these investigations.

JACQUES LESPÉRANCE, M.D.

BOOKS RECEIVED

ROENTGENOLOGY OF INTRACRANIAL MENINGIOMAS. By Sidney P. Traub, M.D., Associate Professor of Radiology, University of Saskatchewan College of Medicine; Assistant Director of Radiology, University Hospital, Saskatoon, Canada; Honorary Consultant Radiologist, Saskatchewan Cancer Commission; and formerly, Fellow in Neuroradiology, Montreal Neurological Institute, Montreal, Canada. Cloth. Price, $14.00. Pp. 238, with 128 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

DISEASES OF THE INTERVERTEBRAL DISC AND ITS SURROUNDING TISSUES. By Reuben Rabinovitch, B.A., M.Sc., M.D., Assistant Professor in Neurology and Neurosurgery, McGill University, Faculty of Medicine, Montreal; and Assistant Neurologist, The Department of Neurology and Neurosurgery, The Royal Victoria Hospital and The Montreal Neurological Institute. Cloth. Price, $8.50. Pp. 152, with 78 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

RADIOACTIVITY IN MAN; A SYMPOSIUM HELD AT THE VANDERBILT UNIVERSITY SCHOOL OF MEDICINE. Edited by George R. Meneely, M.D. Cloth. Price, $16.50. Pp. 491, with numerous illustrations and tables. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

THE PATHOLOGY OF IONIZING RADIATION. By Shields Warren, M.D., Sc.D., LL.D., Professor of Pathology, Harvard Medical School at the New England Deaconess Hospital; Pathologist, New England Deaconess and New England Baptist Hospitals, Boston, Mass. Cloth. Price, $3.00. Pp. 42, with 17 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

A MANUAL OF CUTANEOUS MEDICINE. By Donald M. Pillsbury, M.A., D.Sc. (Hon.), M.D., F.A.C.P., Professor and Chairman of the Department of Dermatology, University of Pennsylvania School of Medicine; Director, Commission on Cutaneous Diseases, Armed Forces Epidemiological Board; and President, XII International Congress of Dermatology; Walter B. Shelley, M.D., Ph.D., F.A.C.P., Professor of Dermatology, University of Pennsylvania School of Medicine; and National Consultant in Dermatology to the Surgeon General, U. S. Army and U. S. Air Force; and Albert M. Kligman, M.D., Ph.D., Professor of Dermatology, University of Pennsylvania School of Medicine; and Professor of Dermatology, University of Pennsylvania Graduate School of Medicine. Cloth. Pp. 430, with 234 illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pa., 1961.

DAS CARCINOMA COLLI UTERI, SEINE PROGNOSE UND BEHANDLUNG; MIT BESONDERER BERÜCKSICHTIGUNG DER STRAHLENTHERAPIE. By Dr. med. Wilhelmine Janisch-Raskovic, ehe. Strahlentherapeutin der vorm. Staatl. Frauenklinik Chemnitz jetzt Städt. Frauenklinik Karl-Marx-Stadt. Cloth. Pp. 649, with many illustrations and tables. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961.

MEDICAL ALMANAC 1961–62. Compiled by Peter S. Nagan, A.B., M.A., M.S. Paper. Pp. 528. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pa., 1961.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Deauville Hotel, Miami Beach, Fla., Sept. 26–29, 1961.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Charles G. Stetson, 350 Engle Street, Englewood, N. J. Annual meeting: Waldorf-Astoria Hotel, New York, N. Y., April 1–4, 1962.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb.
Treasurer, Dwight Vincent Needham, 713 E. Genessee St., Syracuse, N. Y. Annual Meeting: Palmer House, Chicago, Ill., Nov. 26–Dec. 1, 1961.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Roosevelt Hotel, New York, N. Y. Feb. 7–10, 1962.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: New York City, June 26–30, 1961.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building. Rochester, Minn. The Fall 1961 examination will be held at the Shoreham Hotel, Washington, D. C., December 4–7, inclusive; the deadline for filing applications is July 1, 1961. A Special Examination in Nuclear Medicine will be offered if there are sufficient applications.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

TENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

SEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in São Paulo, Brazil, September 3–10, 1961.
Secretary-General, Dr. Walter Bomfim-Pontes, Rua Cesario Motta, No. 112, São Paulo.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. Don E. Matthieson, 926 East McDowell Rd., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. B. Scruggs, Arkansas Baptist Hospital, Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary, Dr. Melvin M. Figley, Department of Radiology, University of Washington, Seattle 5, Wash. Annual meeting to be announced.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

BLOCKLY RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St., Philadelphia, Pa.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph Arcomano, 168 Clinton St., Brooklyn 1, N. Y. Meets first Thursday of each month October through May.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Kenneth H. Seagrave, 537 Delaware Ave., Buffalo 2, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Robert L. Freidman, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. William F. Hutson, 5145 N. California Ave., Chicago, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Norman E. Berman, 14404 S. Park Blvd., Shaker Hgts. 20, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. Seward Imes, 1845 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. James L. Krieger, 85 Jefferson St., Hartford, Conn. Meets first Friday in February and April.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary, Dr. F. J. Bonte, 5201 Harry Hines Blvd., Dallas 35, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. Marsh Frere, Jr., 205 Medica Arts Building, Knoxville, Tenn. Meets in January and September.

EASTERN CONFERENCE OF RADIOLOGY
Secretary, Arrangements Committee, Dr. Philip Myers, Baltimore City Hospital, Baltimore 24, Md. Annual meeting to be announced.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

EASTERN RADIOLOGICAL SOCIETY
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C. Meets at Mid Pines Club, Southern Pines, N. C., April 29–May 2, 1962.

FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred G. Levin, 837 DuPont Bldg., Miami, Fla. Meets twice annually, in the spring with the annual State Society Meeting, and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph C. Rush, 1800 Druid Rd., Clearwater, Fla.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary, Dr. Carl E. Balli, 907–8 Huntington Medical Building, Miami 32, Fla. Meets monthly third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. William E. Powers, St. Louis, Mo.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. John Douglas Reeve, Texas Medical Center Library, Jesse H. Jones Library Bldg., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

IDAHO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the Spring and Fall.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in spring and fall.

INDIANA ROENTGEN SOCIETY, INC.
Secretary, Dr. David E. Wheeler, 1500 North Ritter, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Roger K. Wallace, Riley County Hospital, Manhattan, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. Lawrence A. Davis, 226 East Chestnut St., Louisville, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Abraham Berens, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Stilson, 1720 Brooklyn Ave., Los Angeles, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles.

MAINE RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Poulin, Thayer Hospital, Waterville, Maine. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

MEMPHIS ROENTGEN SOCIETY
Secretary, Dr. Hollis H. Halford, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

MISSISSIPPI RADIOLOGICAL SOCIETY
Secretary, Dr. Jack K. Goodrich, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

MONTANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. K. Boughn, 35 11th Ave., Helena, Montana. Meets at least once a year.

NASSAU RADIOLOGICAL SOCIETY
Secretary, Dr. Alan E. Baum, 100 Nowbridge Rd., Hicksville, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Ronald E. Waggener, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Robert E. Wise, 605 Commonwealth Ave., Boston 15, Mass. Meets third Friday of each month, October through May at The Longwood Towers, Brookline, Mass.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Bernard Wolf, Mt. Sinai Hospital, New York, N. Y. Meets monthly on third Monday, New York Academy of Medicine at 4:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. A. B. Croom, 624 Quaker Lane, High Point, N. C. Meets in the spring and fall each year.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. R. F. Raasch, Post Office Box 990, Dickinson, North Dakota. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Lester I. Citrin, St. Mary's Hospital, Troy, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Paul D. Meyer, 125 S. Grant Ave., Columbus, Ohio. Annual meeting to be announced.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. E. D Greenberger, Medical Arts Bldg., McAlester, Okla. Meets in January, May and October.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. George R. Satterwhite, 1123 S.W. Yamhill, Portland, Ore. Meets monthly from October to June on

the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. John N. Burkey, 555 Dental Bldg., Seattle, Wash. Annual meeting to be announced.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Frederick R. Gilmore, 234 State St., Harrisburg, Pa. Annual meeting to be announced.

Philadelphia Roentgen Ray Society
Secretary, Dr. Robert B. Funch, Department of Radiology, Germantown Hospital, Philadelphia 44, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Ross H. Smith, St. Margaret Memorial Hospital, Forty-Sixth St., Pittsburgh 1, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. Donald Janny, Cincinnati, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Philip S. Arthur, 274 Young Hotel Bldg., Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Robyn Hardy, 4324 Magnolia St., New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Austin J. Tidaback, 912 Prospect Ave., Plainfield, N. J. Meets at Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Robert H. Greenlaw, 260 Crittenden Blvd., Rochester 20, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 10–12, 1961.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Wayne P. Whitcomb, Hospital of St. Raphael, New Haven, Conn. Meetings are held bimonthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1746 K St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary. Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. Richard G. Lester, 412 Union St., S.E., Minneapolis 14, Minn. Annual meeting: Deaville Hotel, Miami Beach, Fla., Sept. 25, 1961.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. *Administrator*, Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Ralph S. Clayton, 1501 Arizona, Bldg. 2-A, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Annual meeting: Austin, Texas, Jan. 19 and 20, 1962.

Tri-State Radiological Society
Secretary, Dr. John Marchand, 118 S.E. First St., Evansville, Ind. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Powell G. Dillard, Jr., 715 Church Street, Lynchburg, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Joseph T. Houk, 14303 Ambaum Blvd., Seattle 66, Wash. Meets third Monday of each month from September through April at the University of Washington Medical School.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Anthony A. Maglione, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2-37, zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15. México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César E. Rosa-Perez, Fondo del Segura del Estado, Parada 1, San Juan 8, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Edmonton and District Radiological Society
Secretary, Dr. S. C. Windle, 105 Northgate Bldg., Edmonton, Alberta. Meets first Tuesday of each month, Oct. to May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Annual meeting: Marlborough Hotel, Winnipeg, Man., Jan. 22–24, 1962.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, D. Camillo Segreto, Avenida Angélica, 1.170, Caixa Postal 5984, São, Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Annual meeting to be announced.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. LEUCUTIA, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Brannon, Earl W. Congenital malformation of the atlanto-axial joint with dislocation. *J. Bone & Joint Surg.*, Dec., 1960, *42A*, 1377–1380. (From: Department of Orthopaedic Surgery, United States Air Force Hospital, Lackland, San Antonio, Texas.)

The author discusses the unique anatomy and function of the first two cervical segments. The causes of dislocation at this level, with emphasis on the effects of dysplasia of the dens, are reviewed. In a case report he describes the clinical findings and treatment.

The annular atlas, supporting the head and surrounding the odontoid process, articulates with the axis in a manner which permits rotation at the atlantoaxial joint. The efficiency of the transverse and check ligaments in maintaining stability is dependent upon the integrity and normal development of the osseous elements at this level; because of the intimate structural relationship to the spinal cord, minor luxations may produce neurologic abnormalities.

Dislocation may result: (1) when the anterior or posterior arch of the atlas is fractured; (2) with fracture of the odontoid process; (3) as a result of demineralization of bone with resultant disruption of ligamentous attachments, as might be caused by infection; and (4) due to congenital absence or hypoplasia of the odontoid process. With hypoplasia the defect may be subclinical but the excessive mobility which is produced tends to become progressive, with weakening of other ligamentous attachments and the eventual appearance of neurologic signs.

When instability is the result of hypoplasia of the odontoid process, symptoms may first be noted in the third decade. They consist of pain in the neck, shoulders and occipital region, which is aggravated by lifting, and there may be associated numbness and paresthesia of the arms. Physical signs may consist of some limitation of neck extension and induction of the pain pattern when the neck is flexed. The spinous process of the axis is prominent. Deep tendon reflexes in all extremities may be hyperactive.

In the case cited, roentgenograms showed a malformed and shortened odontoid process with forward dislocation of the occiput and atlas; dislocation was exaggerated upon flexion. Spine fusion was performed from the occiput to the fifth cervical vertebra. At operation, partial absence of the laminae, a bifid axis and a lack of development of the ligaments overlying the dura were demonstrated. The surgical treatment was successful.—*Jack Reynolds, M.D.*

Luccarelli, S., and De Ferrari, U. Studio clinico-radiologico di un caso di origine anomala (dall'a. carotide esterna) dell'arteria vertebrale sinistra. (Clinical and radiologic study of a case of anomalous origin [from the external carotid artery] of the left vertebral artery.) *Radiol. med.*, Oct., 1960, *46*, 963–974. (From: Divisione di Neuro-Psicopatologia and Istituto di Radiologia, Ospedale Maggiore di Milano, Milano, Italy.)

The anomaly described is one of origin and of course of the left vertebral artery. It was casually discovered in an angiographic examination. The presence of such a singular vascular malformation was not suspected clinically.

The angiographic examination was carried out as follows: The contrast medium was injected into the left carotid artery at the level of the common trunk. The internal carotid artery was visualized normally and, at the same time, to the authors' surprise, vessels of the vertebral basilar system were opacified.

It was determined surgically and roentgenologically that the left external carotid, after having given origin to the superior thyroid, lingual and maxillary arteries, continued cephalad, assuming the morphologic and functional characteristics of the vertebral artery and coursing totally outside the canal formed by the transverse foramina.

The authors give an excellent summary of the embryology and anatomy of the anomalies of the vertebral artery.

Clinically, their patient, a hypertensive female fifty-one years of age, presented the picture attributed to softening of the posterior sylvian portion of the left cerebral hemisphere.—*Anthony A. Blasi, M.D.*

Ecker, Arthur, and Perl, Theodore. Percutaneous injection of the thalamus in Parkinsonism; a preliminary report: relief of bilateral facial grimaces. *A.M.A. Arch. Neurol.*, Sept., 1960, *3*, 271–278. (From: Departments of Surgery [Neurosurgery] and of Radiology, State University of New York, Upstate Medical Center, Syracuse, N. Y.)

Injection of the thalamus using the well-known cannula and balloon technique results in a mortality of 2.4 per cent and an additional complication rate, usually hemiplegia, of 3 per cent. With this new method, the target in the ventrolateral nucleus of the thalamus is determined by measuring from the calcified pineal body. If it is not calcified, a pneumoencephalogram is required for localization of the pineal body, the target is aligned through the center of the foramen ovale to the skin, thus indicating the point of entry. A 21 gauge guide needle is inserted through the skin to the foramen ovale. Then a longer 26 gauge needle is passed a measured distance through the guide needle to the target, after which 0.2 cc. of 1 per cent procaine or lidocaine is injected through the inner needle to determine the clinical

effect. Finally, small amounts of absolute alcohol (0.1 to 0.2 cc.) are injected to obtain a permanent effect.

The target area is the most anteroinferolateral portion of the thalamus. The target lies 16 mm. lateral to the midline, and 10 mm. in front of the posterior commissure in a horizontal plane 2 mm. above that which includes the anterior and posterior commissures. There is usually dramatic relief of rigidity and/or tremor within seconds. If the symptoms recur after a few minutes, the injection is repeated. When the maximum benefit is obtained, the procedure is concluded by withdrawing the needle.

Seven patients were injected using this method. In all cases contralateral rigidity and tremor were largely or completely relieved.—*Edwin F. Koch, Jr., M.D.*

NECK AND CHEST

CATHCART, RICHARD T., THEODOS, PETER A., and FRAIMOW, WILLIAM. Anthracosilicosis; selected aspects related to the evaluation of disability, cavitation, and the unusual x-ray. *A.M.A. Arch. Int. Med.*, Sept., 1960, *106*, 368–377. (From: Barton Memorial Division, Jefferson Medical College Hospital, Philadelphia, Pa.)

The authors present their findings in regard to selected aspects of the pneumoconiosis problem in anthracite coal workers based on a ten year study of some 1,690 long-term workers.

They found that the degree of anthracosilicosis demonstrated roentgenographically did not necessarily parallel ventilatory function. Ventilatory function and, therefore, the degree of disability appeared to be much more closely related to the degree of concomitant emphysema.

It is well known that tuberculosis is a not infrequent complication of anthracosilicosis and that it is a frequent cause of cavitary disease. None the less, cavitary disease without tuberculosis does occur in anthracosilicosis and this possibility should be recognized. Of 182 miners with cavitary disease, in 18 per cent it was due not to tuberculosis but to ischemic necrosis within conglomerate masses of nodules.

The authors present cases which illustrate the following clinical and roentgen features of ischemic necrosis in anthracosilicosis: (1) cavitation in association with areas of conglomerate fibrosis; (2) sudden and rapid development of a cavity due to liquefaction of a conglomerate area; (3) copious amounts of black sputum; (4) an aseptic clinical course unaccompanied by chills, fever, or leukocytosis; and (5) the fact that this entity does not prognostically influence the course of the underlying anthracosilicosis.

The usual case of anthracosilicosis presents no difficulty in diagnosis. There are numerous cases, however, where the exposure is real and disability is claimed but the roentgen examination does not show the typical silicotic nodules. The roentgen changes may be only increased lung markings and linear fibrosis, mimicking changes frequently seen in men of this age group who have no specific industrial exposure. In such cases lung biopsy is indicated. This procedure has been used in 30 cases and has been most helpful in establishing the presence or absence of silicosis. In 4 of 19 miners with negative roentgenograms, silicotic nodules were found. In 5 of 11 miners with the "linear fibrosis" pattern, evidence of silicosis was found.—*Martin A. Thomas, Major, USAF MC*

MACFADYEN, D. J. Wegener's granulomatosis with discrete lung lesions and peripheral neuritis. *Canad. M.A.J.*, Oct., 1960, *83*, 760–764. (From: Department of Medicine, University Hospital, Saskatoon, Saskatchewan, Canada.)

Wegener's granulomatosis comes within that spectrum of disease states ranging from necrotizing and granulomatous processes without vasculitis to vasculitis without granulomata. The granulomatous processes are represented by such diseases as midline lethal granuloma, wherein generalized vascular lesions are usually absent. The group of vasculitis processes without granulomata includes hypersensitivity angiitis.

Within these two extremes lies Wegener's granulomatosis, along with other mixed forms such as allergic angiitis with granulomatosis. Wegener's granulomatosis is distinguished from other members of this group by the predominance of respiratory symptoms and lesions.

The author presents an interesting case of Wegener's granulomatosis. This case featured well-circumscribed granulomata which were similar roentgenographically to metastatic lesions. (One series of such cases demonstrated dense, circular or oval opacities in the lungs in slightly more than 50 per cent of the patients.) The case presented also showed a predominance of vasculitis symptoms but a paucity of respiratory symptoms. The peripheral neuritis which was present closely resembled that found in cases of periarteritis nodosa.—*Charles H. Helmen, M.D.*

MELOT, G., and BOLLAERT, A. Tentative de mesure de la circulation pulmonaire par l'angiopneumographie. (An attempt to measure pulmonary circulation by angiopneumography.) *J. belge de radiol.*, May, 1960, *43*, 445–478. (From: Service de Radiodiagnostic de l'Hôpital universitaire Saint-Pierre, Bruxelles, Belgium.)

In order to understand the pulmonary circulatory function, it is necessary to study the extent of the pulmonary vascular bed, the caliber of the pulmo-

nary vessels and the rate of intrapulmonary circulation. This report is based on angiopneumographies safely performed on 450 patients with various pulmonary diseases. The results were supported by bronchospirometric findings in most cases.

Studies of the caliber of the vessels established the following normal values, which are comparable to those reported by Dotter and Steinberg: pulmonary artery, 32 mm.; left branch, 20 mm.; right branch, 22 mm.; lobar artery, 12 mm.; segmental artery, 7.5 mm.; subsegmental artery, 6 mm.; peripheral, 1–2 mm.; pulmonary veins, 12 mm.

Circulation time, as determined by the time needed for opacification of the pulmonary artery and its corresponding vein, is 2–3 seconds. The duration of the arterial, capillary and venous phases of opacification is 3 seconds with a maximum of 4½ seconds.

The authors report in detail 7 cases illustrating striking pathologic findings. Case I: long-standing tuberculosis of the right upper lobe with accompanying collapse, hydrothorax, and retraction of the heart and mediastinum to the left. Angiopneumography revealed markedly increased vascularity of the right lung with some increase in caliber, despite retraction of the pulmonary artery to the left. There was nonvascularization of the left lung. Case II: a history of left pneumonectomy for bronchogenic carcinoma. Angiopneumography demonstrated marked hypervascularization of the right lung. Case III: thoracoplasty with resection of the six left upper ribs and accompanying lung collapse. Angiopneumography revealed decreased vascularity of the left lung with well-developed right pulmonary vascularization. Case IV: left hilar carcinoma and left upper lobe atelectasis. Angiopneumography showed decreased aeration and decreased vascularization of the left lung but normal vascularization of the right lung. Case V: a history of left hydropneumothorax with almost complete collapse of the left lung and marked residual pleural thickening and retraction of the heart and mediastinum to the left. Angiopneumography visualized markedly decreased vascularity of the left lung and normal vascularity in the right lung. Case VI: carcinoma of the right main bronchus, collapse of the right lung, and retraction of the heart and mediastinum to the right. There was no ventilation of the right lung and markedly decreased vascularity was demonstrated by angiopneumography; the vascular bed on the left side was normal. Case VII: generalized pulmonary emphysema of long standing. Angiopneumography revealed markedly decreased vascularity of the pulmonary bed with decreased caliber of the vessels; however, there was dilatation of the proximal or larger vessels, bilaterally.

The authors find that in significant unilateral lesions (tuberculosis, cancer, pyosclerosis) there is a progressive decrease in the caliber of the arteries and veins on the affected side, whereas on the healthy or contralateral side the vessels are larger than normal. In pulmonary emphysema, however, the appearance is quite striking in that the larger (proximal) vessels are abnormally dilated, while the peripheral vessels are decreased both in number and caliber.

Bronchospirometric studies confirm the fact that oxygen consumption in the lung is increased with increased vascularity and decreased with decreased vascularity, with an accompanying decrease in the caliber of the vessels and the rate of flow. Thus, with pneumography it is possible to determine the functional capacity of a diseased lung and the extent to which the normal lung is able to compensate. This information is particularly useful when surgical intervention is being considered.—*William H. Shehadi, M.D.*

Starer, F., and Sutton, David. Aortic occlusion (Leriche's syndrome) in mitral stenosis; report of six cases. *Brit. M. J.*, Aug., 1960, 2, 644–647. (From: Radiological Department, St. Mary's Hospital, London, England.)

Peripheral emboli are a frequent hazard to the patient with mitral stenosis and auricular fibrillation. The insidious onset of aortic thrombosis in patients with mitral stenosis has not been previously described. The authors report 6 cases of gradual aortic occlusion in patients with mitral stenosis and auricular fibrillation. In fact, 10 per cent of their cases with aortic thrombosis occurred in patients with mitral stenosis. In all of the authors' cases, repeated peripheral emboli preceded aortic thrombosis.

Two theories of the pathogenesis of aortic thrombosis in this group of patients are given. The first is that an extensive thrombosis involving the aorta was built up by the accumulation of repeated small emboli, most of which were too small to produce major symptoms by themselves. The second possibility is that there was progressive clotting in the vessel proximal to the peripheral embolic occlusion. On present evidence it is not possible to be certain which of the views discussed is correct.—*Eugene C. Klatte, M.D.*

Arcilla, Rene A., and Gasul, Benjamin M. Congenital dextrocardia; clinical, angiocardiographic, and autopsy studies on 50 patients. *J. Pediat.*, Jan., 1961, *58*, 39–58. (From: The Pediatric Cardiophysiology Department, Cook County Children's Hospital and Hektoen Institute for Medical Research, Chicago, Ill.)

Dextrocardia is associated sufficiently often with cardiac anomalies amenable to palliative or corrective surgery to inspire more than academic interest in the subject. The cases previously described as dextrocardia without situs inversus, and now classified as dextroversion complex or mixed dextrocardia, are almost always accompanied by severe congenital malformation of the heart. Dextrocardia secondary to anomalies of the lungs, diaphragm or chest (ex-

trinsic dextrocardia) usually has no associated cardiac abnormality.

Data on 55 cases of congenital dextrocardia were reviewed. Five were excluded because of incomplete records. (Cases of extrinsic dextrocardia were not considered.) There were 26 males and 24 females, and the ages ranged from birth to twelve years with 1 exception. Each of these patients was followed to surgery and, in 40 per cent, to autopsy.

The degree of dextrocardia was determined by the angle formed by the longitudinal axis of the heart with the midsagittal axis of the chest in the horizontal plane. This is normally 40° to the left. An angle of 30 to 40° to the right was considered to be complete dextrocardia and from 0 to 20°, incomplete. This reflects the degree of pivotal rotation of the heart along the horizontal plane in the extrinsic group, and the degree of cardiac heterotaxy in the intrinsic group.

The cases were classified into five categories:

Type I. Mirror-Image Dextrocardia (anatomic inversion of the chambers). Six of the 11 cases in this group had associated cardiac anomalies. This is at variance with other reported series, the majority of which record normal cardiac findings in this type of dextrocardia. The discrepancy is probably due to a combination of incorrect classification (several cases reported are clearly incomplete dextroversion or mesoversion) and differences in the age groups studied (those with serious cardiac anomalies die in infancy).

Type II. Dextroversion Complex (complete and incomplete mesoversion). Fourteen of the 15 cases in this group had associated cardiac anomalies. In complete dextroversion the auricles and ventricles, and with them the greater vessels, are reversed in position in the sagittal plane as if the heart had undergone 90° pivotal rotation about a posteriorly placed vertical axis. Mesoversion is similar but with less than 90° rotation so that the heart appears narrower than normal in the posteroanterior view and the relative position of the chambers is the same as ordinarily seen in a left anterior oblique view.

Type III. Mixed Dextrocardia. Either the atria alone may be transposed, or the ventricles alone, with or without transposition of the great vessels. The identification of either atrium by contrast roentgenography alone is not possible but, when a superior vena cava enters a venous atrium on the left side, atrial inversion is probably present. If there is no associated situs inversus, mixed dextrocardia should be suspected. The 4 cases of this type all had associated anomalies.

Type IV. Congenital Dextroposition. The position is the same as that which the heart would assume if it were shifted *en masse* to the right. Two of the 3 cases of this type had associated cardiac anomalies.

Type V. Extrinsic Dextrocardia. Only 4 of 17 cases showed associated anomalies.—*Lois Cowan Collins, M.D.*

ARCILLA, RENE A., and GASUL, BENJAMIN M. Congenital dextrocardia; clinical, angiocardiographic, and autopsy studies on 50 patients. *J. Pediat.*, Feb., 1961, *58*, 251–262. (Address: Pediatric Cardiology, Cook County Children's Hospital, Chicago, Ill.)

The electrocardiographic studies in the 50 cases of congenital dextrocardia reported in the article abstracted immediately above are described for each of the five types of dextrocardia into which these cases were classified. No typical electrocardiographic findings were encountered in Type IV and the findings in these cases were normal. In the other cases the variations could be explained in most instances by the abnormal position and location of the cardiac chambers.

The roentgenologic findings in Types I, II, and III are similar in most respects, consisting of an elevated left diaphragm and identical cardiac configurations in the posteroanterior and oblique views which are the reverse of normal.

A right aortic arch is common in Type I and situs inversus always accompanies this type. In Type IV dextrocardia, the heart shadow is medially located in the frontal view and assumes normal configuration in the oblique views.—*Lois Cowan Collins, M.D.*

LANG, ERICH K., and SABISTON, DAVID C., JR. Coronary arteriography in the selection of patients for surgery. *Radiology*, Jan., 1961, *76*, 32–38. (Address: The Johns Hopkins Hospital, Baltimore 5, Md.)

Coronary arteriography is primarily employed to determine the site and extent of arterial occlusion in the selection of patients for coronary endarterectomy. A series of 63 examinations has been evaluated. A so-called flushing technique was used in 40 examinations with satisfactory results in 32. Examinations utilizing the Seldinger technique (10 cases) and flushing with acetylcholine-induced arrest (5 cases) were unsatisfactory for the most part. In 8 cases, and particularly in the cinefluorographic examinations, direct injection into the ostium of the coronary artery has been performed with satisfactory results in all instances.

In the recent past, combined examinations using cinefluorographic techniques and the Schönander film changer have been performed in which the coronary ostium was first selectively engaged and injected for cinefluorography, and then the catheter tip was placed in the coronary cusp and the patient moved to the Schönander film changer for a second injection of contrast material and examination. Comparison of the examinations done by these two methods strongly supports the opinion that both cinefluorography and large detail film are necessary for the correct diagnosis. Electrocardiographic monitoring during the entire procedure is regarded as imperative.

Of the 41 patients examined by coronary arteriography in this series, localized lesions (amenable to endarterectomy) were present in 9, multiple lesions and peripheral lesions (generally unsuitable for endarterectomy) in 26, and a normal coronary artery in 6. Of the 6 patients operated upon (including an endarterectomy with removal of the obstruction), 5 showed good postoperative results and 1 died in the immediate postoperative period. The finding of normal coronary arteriograms in 6 patients with equivocal electrocardiographic tracing and negative Master's exercise test ruled out organic heart disease, thus helping to eliminate from surgical consideration a group of normal individuals who presented a clinical history of angina pectoris.—*Walter H. Jarvis, Jr., M.D.*

Rossetti, M. Reflux oesophagitis and bleeding oesophageal varices. *German Med. Monthly*, Jan., 1961, *6*, 1–3. (From: Department of Surgery, University of Basle, Basle, Switzerland.)

The simultaneous occurrence of esophageal varices and insufficiency of the cardia with gastroesophageal reflux is reported in 3 cases. Reflux esophagitis may cause or favor hemorrhage from the varices.

In Case I there were "idiopathic" varices and sliding hernia; in Case II "idiopathic" varices were associated with malformation of the cardia and gastric fundus; and in Case III varices secondary to portal vein thrombosis were combined with malformation of the cardia and fundus.

In each case hemorrhage from the varices occurred. In the first case transabdominal plication of the gastric fundus and under running of the venous plexus stopped the bleeding. In the other 2 cases it was necessary to perform an esophagogastrectomy.—*James J. McCort, M.D.*

Abdomen

Fournier, M., and Guien, Claude. Les stases duodénales. (Duodenal stasis.) *J. de radiol., d'électrol et de méd. nucléaire*, Dec., 1960, *41*, 757–768. (From: Service central de Radiologie de l'Hôpital de la Conception, Marseille, France.)

Duodenal stasis is more likely to occur in tall, slender, hyposthenic individuals. It appears usually between the duodenal cap and the knee of the duodenum. Concomitant findings are slight dilatation, alteration of the mucosal pattern, changes in contour and dyskinesia. The patient usually complains of a sensation of "weight" or heaviness and the feeling that the noon meal is still in the stomach long after he has eaten. This sensation disappears with borborygmus as the patient maneuvers into a position he knows will give him relief.

The roentgen signs are: (1) modification of the caliber of the duodenum, (the most revealing sign—in 1 extreme case the caliber was 9 cm.), with varying degrees of dilatation; and (2) modification of the mucosal pattern with flattening of the folds or almost total effacement; sometimes the folds present a polyhedral pattern.

The cause of stasis may be functional, organofunctional, organic or idiopathic. Functional causes may be disturbances of innervation or of muscular tonicity. The use of certain drugs is associated with disturbances of motor function; morphine causes gastric hyperperistalsis and duodenal stasis, while atropine relieves pyloric spasm and causes dilatation of the duodenum. Endocrine disturbances, particularly hypothyroidism, may be associated with stasis, which regresses under treatment. Vagotonia as a result of the Dragstedt operation may be a significant factor. Avitaminosis and acid-alkali imbalance are also mentioned as causes.

Organofunctional stasis may occur after gastrectomy or gastroenterostomy, and it may accompany gastric or duodenal ulcers, duodenal diverticulosis, and other abdominal diseases such as cholecystitis and pancreatitis. Organic stasis may occur with complete or partial duodenal obstruction. It may result from incomplete obstruction due to congenital atresia or an annular pancreas caused by ectopic pancreatic tissue. Stasis also occurs in a so-called mobile duodenum, or from extensive pressure by mesenteric vessels, neighboring organs (pancreas, kidney, biliary tract, colon, stomach), retroperitoneal neoplastic masses as well as intrinsic lesions of the descending duodenum (carcinoma, especially of the region of the ampulla, benign tumors) and foreign bodies.

Essential or idiopathic stasis with no demonstrable cause is a diagnosis that can only be made after ruling out all other causes.—*William H. Shehadi, M.D.*

Chocholáč, Joseph. (Prague, Czechoslovakia.) Les modifications de l'activité motrice de l'intestin grêle dues à une perturbation de l'afflux de la bile dans le duodénum. (Modifications in the motor activity of the small intestine due to a disturbance of the flow of bile into the duodenum.) *J. de radiol., d'électrol. et de méd. nucléaire*, Dec., 1960, *41*, 769–778.

Disturbances in the flow of bile into the small intestine may result from: (1) deficiency in the excretory function of the liver due to hepatic cellular degeneration (hepatitis, cirrhosis, ascending cholangitis, malignant disease) or to cholestasis, either primary or secondary to extrahepatic obstruction; and (2) obstruction of flow through the biliary ducts by calculi, inflammatory diseases of the biliary tract, and tumors of the biliary tract or of adjacent organs, especially the pancreas.

The author mentions the conflicting opinions

expressed in the literature on the effect of bile on intestinal motility. He also refers to the work of investigators who have demonstrated experimentally that insufficiency of bile in the digestive tract causes slow emptying of the stomach and slow transit through the intestines.

The author has studied the roentgen appearance of the small intestine in 132 patients with biliary tract disease. When an organic lesion of the stomach or duodenum was suspected, a duodenal intubation was performed to assure the passage of barium into the small intestine. In 72 per cent of these patients, the changes in the small intestine were: a decrease or lack of tonus, especially in the jejunum; dilatation of the lumen; continuity of the barium column; and suppression of peristaltic activity at the level of the involved segments. There was atony and over-all delayed transit through the small intestine.

After administering a barium meal to patients who presented the above findings, the author introduced 15 to 20 cc. of bile (8–10 per cent fel tauri) via a duodenal catheter. Within fifteen minutes, there was peristaltic activity and increased tonus of the intestines, with rapid progress of the intestinal contents.—*William H. Shehadi, M.D.*

Delannoy, E., and Paris, J. A propos d'un cas nouveau de schwannome isolé du rectum. (Concerning a new case of isolated schwannoma of the rectum.) *Presse méd.*, May 25, 1960, *68*, 1021–1022. (From: Clinique Chirurgicale des Adultes, Hôpital Régional Cité Hospitalière, Bloc Est, Lille, France.)

A sixty-eight year old patient, who had had constipation, rectal tenesmus and frequent mucous discharge over a two year period, suddenly experienced acute abdominal pain. At rectal examination, a tender, firm mass was found in the sacral concavity and bulging into the posterior wall of the rectum. At rectoscopy the mucosa was smooth and bright red in color, without ulceration or bleeding.

The clinical diagnosis was retrorectal abscess, since there was a concomitant hyperthermia which responded to antibiotic therapy. A similar attack of pain and fever occurred eleven months later and was followed by several recurrences.

By retroanal incision and resection of part of the sacrum, a tumor was removed from the rectal wall. It was easily freed from the mucosa and macroscopically resembled a fibrosarcoma.

Histologic examination demonstrated typical aspects of neurinoma with some cellular and nuclear atypical findings which indicated some possibility of malignant degeneration. No recurrence was present when the patient died fourteen months later from myocardial infarct.

This is the thirteenth such case recorded in the medical literature.—*Guy Albot, M.D.*

Mosca, Lidio G., de Di Rienzo, Nelida Vanetta, Scavino, Giovanni M., and Croppi, Carlos J. (Cordóba, Argentina.) La colecisto y la colangiografía por via oral con un nuevo medio de contraste. (Oral cholecystography and cholangiography with a new contrast medium.) *Radiología, Panamá*, Dec., 1960, *11*, 7–20.

A series of 100 cases was examined using a new contrast medium, acid beta-(3-dimethylamino-methylamino 2,4,6-triiodo phenyl)-propionate. It has an iodine content of 63.7 per cent. In most cases, six capsules of 500 mg. each were administered sixty minutes before roentgenography was done, at which time the gallbladder usually was opacified. For cholecysto-cholangiography, six capsules were administered twelve hours before the examination. If visualization was not obtained at this time, an additional six capsules were administered and the patient was re-examined in three hours. Occasionally, it was necessary to make additional roentgenograms at four and five hours after the administration of the last dose.

For cholangiography in cholecystectomized patients, six capsules were given the evening before and twelve capsules three hours before the examination. If the common duct was not visualized, additional roentgenograms were made five hours after the last dose had been given.

Positive cholecystograms were obtained in 69 per cent of the cases examined. In 66 per cent of the patients with positive gallbladders, cholangiograms were obtained. Of the cholecystectomized patients, 42 per cent also showed positive cholangiograms. Of 21 cases with nonvisualized gallbladders when they were examined with ordinary triiodized contrast medium, 24 per cent were positive when the new medium was used.

The advantages of this new contrast medium include perfect tolerance, no residual intestinal opacities, an increased percentage of visualized gallbladders and successful cholangiography even in cholecystectomized patients.—*Murray M. Friedman, M.D.*

Franke, W. Beitrag zur röntgenologischen Darstellung einer seltenen Anomalie der Gallenwege und zum Problem der postoperativen Cholangitis. (Contribution to the roentgenologic demonstration of a rare anomaly of the bile ducts and the problem of postoperative cholangitis.) *Chirurg*, Jan., 1961, *32*, 14–19. (From: Medizinische Klinik des St. Johannes-Hospital, Dortmund, Germany.)

A rare anomaly of the bile ducts is described. A secondary branch (ventral) of the right hepatic duct

communicated directly with the common duct, and the cystic duct connected with this abnormal branch. It was possible to demonstrate this anomaly because of a fistula between the pylorus and gallbladder due to a gallstone perforation with a marked stenosis of the distal end of the common duct. It is believed that this is the first case in which the anomaly has been demonstrated in a living patient.

The author does not share the optimism of some surgeons as to the long-term results of a choledochoduodenostomy. It seems almost unavoidable that there will be considerable reflux and stasis of duodenal secretion into the biliary ducts, which ultimately will lead to cholangitis. Careful roentgen examinations after choledochoduodenostomy would probably show abnormal biliary ducts more often than is now suspected.—*Hans W. Hefke, M.D.*

Grill, W. Indikation und Technik der intraoperativen Gallengangsrevision. (Indication and technique for the operative bile duct revision.) *Chirurg*, Jan., 1961, *32*, 1–9. (From: Chirurgische Universitätsklinik, München, Germany.)

The postcholecystectomy syndrome is present in 10–30 per cent of patients, depending on the evaluation of symptoms. The reason is either an erroneous diagnosis or incorrect evaluation of operative findings or technique.

Prevention of recurrent symptoms depends mainly on finding all the stones which may be present in the bile ducts or on recognizing an existing stenosis of the papilla. Stones in the ducts are palpable only in about one-half of the cases. Stenosis of the papilla cannot be palpated. Probing of the ducts fails, in two-thirds of the cases, to demonstrate stones which are present.

Operative manometry with cholangiography has proved to be the most reliable and least traumatic method for accurately evaluating the condition. The contrast material is used as the manometric fluid; usually three cholangiograms are made, although occasionally a series of them is necessary. In order to avoid changes in the muscle tonus in the biliary ducts, morphine and its derivatives are not used as preoperative medication.

Clinical examples of the method are given with operative cholangiograms. Over-filling of the ducts with contrast material may prevent demonstration of small stones. Air bubbles in the ducts can usually be recognized, but may be mistaken for stones. An erroneous diagnosis can be expected in about 10 per cent of the cases.

Mention is made of papillary stenosis and its diagnosis during operation. Obstruction and deformity of the duct by intrinsic and extrinsic tumors are also discussed.

There is practically no contraindication to this method for evaluation of the bile duct.—*Hans W. Hefke, M.D.*

Albot, G., and Delaviere, Ph. La cholostase mineure et ses variétés (cholostase mineure compensée ou décompensée avec ou sans hépatite satellite, cholostase mineure cirrhogène, cirrhose cholostatique anictérique). (Minor cholestasis and its variants [compensated or decompensated minor cholestasis with or without satellite hepatitis, minor cirrhogenous cholestasis, anicteric cholestatic cirrhosis].) *Semaine hôp. Paris*, Dec. 20, 1960, *58*, 3209–3229.

Recent knowledge of anicteric cholestatic cirrhosis has stimulated the authors to seek the means of making an early diagnosis of minor cholestasis. They believe that the findings from the various diagnostic methods should be considered together.

The earliest, most reliable and most constant signs of minor biliary stasis are histologic (portal sclerosis and Hering's neo-canals). Biologic findings are less constant (hypercholesterolemia, alkaline hyperphosphatasemia, anomalies of the bromsulphalein test). These latter are often slight but their relative increase, considered in conjunction with the clinical course, sometimes gives them a particular diagnostic significance.

The signs of diffuse satellite hepatitis (parenchymatous, mesenchymal or cirrhogenous) may be associated with the preceding signs and include the crossing-through tests or flocculation tests, generalized cell clarification or histologic signs of the cirrhogenous process.

All the signs are dependent on the relative obstruction; removal of the obstruction brings about their progressive disappearance, whether they be of histologic or biologic origin. On the basis of their interrelationship, it is possible to differentiate between compensated or decompensated minor cholestases, occurring singly or in association with a satellite hepatitis.—*Guy Albot, M.D.*

Genitourinary System

Adams, A. Wilfrid. (Bristol, England.) The movable kidney syndrome. A successful nephropexy: historical survey and current assessment. *Brit. J. Surg.*, Nov., 1960, *48*, 319–323.

"The dropped kidney" and its significance remains a matter of debate. Nephroptosis, *per se*, need not connote disease. It is asymptomatic in the majority of cases, but the so-called "movable kidney" may be associated with aching pain in the flank, dysuria and colicky abdominal distress.

The factors responsible for the syndrome are those related to the position of the kidney, the nature of its supporting investments and the amount of torsion or stress on the renal pedicle. Neurovascular elements in the pedicle may undergo stretching with resultant pain. Partial and intermittent ureteral occlusion,

which may occur, would tend to induce pyelectasis.

The author reviews the historic background of the "movable kidney" citing the lectures of Baillie in 1785 and Dietl's description in 1864 of catastrophic renal pain. Nephropexy enjoyed a vogue in the early twentieth century but by the 1930's the plea for conservatism and more careful patient selection was heard. More recently, investigators have sought objective evidence of the significance of stasis and the etiology of the pain.

Diagnosis remains difficult. Urography rarely shows pyelectasis even in the symptomatic patient, and then it is minimal. Some authorities allude to the value of the discovery of delayed excretion and they equate this sign with stasis and, therefore, infection. Functional impairment will be elucidated by advancing cineroentgenography and perfemoral aortography will be helpful in ruling out other causes of nephropathy.

Treatment today, even in cases of the true syndrome, is for the vast majority conservative with a supporting belt and rest being prescribed. Surgery is beneficial in *carefully selected* cases. The author endorses this approach and submits a case report describing a surgical success.—*Jack Reynolds, M.D.*

SPJUT, HARLAN J., and NICOLAI, CHARLES H. The nonvisualizing kidney: a pathologic study of 83 nephrectomy specimens. *J. Urol.*, Jan., 1961, *85*, 115–118. (Address: Surgical Pathology, Barnes Hospital, St. Louis 10, Mo.)

A nonvisualized kidney, according to the arbitrary classification, for the purposes of this paper is defined as one in which there is no visualization of the radiopaque material in the excretory passages of the kidney on roentgenograms made over a period of time up to eight hours. If intravenous urography is not done, the kidney which does not excrete indigo carmine over a period of twenty minutes during cystoscopic examination is considered as being nonvisualized. This term is not interchangeable with nonfunctioning, although, for the purposes of comparison with subsequent pathologic findings, it has been used as such in this discussion.

The authors reviewed the roentgenograms of 255 patients who had had unilateral nephrectomy over a five year period and they studied all the histologic reports and sections from 83 nonvisualized kidneys. A minimum of three histologic sections and the gross description of the removed kidney were used as a crude estimate of potential renal function.

Pyelonephritis was found in 54 of the 83 cases of nonvisualized kidney; 7 instances of renal carcinoma were found; and, in the remaining 22, the lesions included dysplasia, renal artery aneurysm, infarction, arteriolonephrosclerosis, tuberculosis, trauma, and cysts.

Of 13 children under the age of fifteen years, 4 showed pyelonephritis, 5 had evidence of renal dysplasia, and 1 each evidence of Wilms' tumor, granulomatous disease, trauma, or infarction.

Of the 83 nonvisualizing kidneys, 62 had evidence of obstruction of various degrees, 26 contained renal stones, and 22 others were associated with obstructions such as those caused by carcinoma of the bladder, carcinoma of the cervix, and inflammatory or postoperative stenosis of the ureter.

In the group of adults, there were 5 instances where, from the study of the histologic and gross alterations of the removed kidney, the authors estimated there should be 75 per cent or more functional parenchyma. Twelve kidneys were estimated to have at least 50 per cent functional parenchyma and the remaining 66 kidneys had less than 25 per cent functional parenchyma. These estimates are admittedly crude but are correlated with the weight of the kidney and the appearance of the histologic sections.

It is noted that obstruction is an important basic factor in the pathogenesis of a nonvisualizing kidney. In those instances where the kidney is seen to be normal in size at the roentgen examination but fails to function, the authors suggest that one might evaluate the pyelogram as indicating that the kidney has a moderate amount of functional parenchyma.

It is significant that, although 20 per cent of the 83 nonvisualized kidneys did have an abundant amount of normal appearing renal parenchyma, most of these had an obstruction to account for the nonvisualization and no entirely normal kidney was found among the entire group of nonvisualized kidneys.—*George W. Chamberlin, M.D.*

TILLE, D. Non-pathological filling defects of the renal pelvis and calyces. *German Med. Monthly*, Jan., 1961, *6*, 12–13. (From: Central Radiological Institute and the Radiotherapy Department, Municipal Hospital, Berlin-Buch, Germany.)

Three cases of filling defects found in the renal pelvis and calyces are reported. Combined renal percutaneous arteriography and intravenous urography demonstrated that these defects were due to overlying (but not aberrant) renal arterial branches. —*James J. McCort, M.D.*

HEITZMAN, E. ROBERT, and PERCHIK, LAWRENCE. Radiographic features of renal infarction; review of 13 cases. *Radiology*, Jan., 1961, *76*, 39–46. (Address: E. R. Heitzman, 820 S. Crouse Avenue, Syracuse 10, N. Y.)

Thirteen cases of renal infarction are presented by the authors who feel that this infrequently diagnosed condition has definite roentgen features which should lead to its more frequent recognition. Renal artery occlusion is usually the result of thrombosis, trauma, or embolism. Embolism is the most common cause and the usual history is that of abrupt onset of flank

pain in a patient with cardiac or vascular disease. Albuminuria is almost always present and hematuria is fairly common. Acute venous infarction often occurs in septic states and is accompanied by marked toxicity and almost always by hematuria.

The chief roentgen manifestation of acute renal artery occlusion is a nonfunctioning kidney of normal size in conjunction with a normal retrograde pyelogram. Segmental infarction may result in complete nonvisualization, although small infarcts may produce no abnormality or only local failure of calyceal filling. Aortography may then establish the diagnosis. Acute venous infarction also produces nonvisualization with enlargement of the involved kidney as a rule. An old total renal infarction is characterized by nonvisualization at intravenous pyelography, normal retrograde pyelograms except for diminished calyceal size, and small kidney contour.

The authors describe how this condition may be differentiated from other lesions presenting unilateral small kidneys. Recovery following sterile infarction secondary to arterial occlusion is the rule, although the affected kidney atrophies and diminishes in size. Acute venous renal infarction usually results in death unless surgical measures are instituted.—*John S. Alexander, M.D.*

Opit, L. J., McKenna, K. P., and Nairn, D. E. Closed renal injury. *Brit. J. Surg.*, Nov., 1960, *48*, 240–247. (From: University Department of Surgery, Queen Elizabeth Hospital, Woodville, South Australia.)

Two divergent opinions are expressed in the surgical literature concerning the management of renal trauma. The viewpoints of the conservatives and the interventionists are described, along with the belief of the latter that early evaluation by retrograde urography and prompt treatment by nephrectomy result in decreased morbidity and fewer late sequelae.

The authors, of conservative persuasion, review the diagnosis, management and course of 77 cases of closed renal injury in order to determine the frequency of early and late complications. Those conservatively managed patients whose clinical or pyelographic findings during the acute course could have prompted surgical intervention were closely studied. An estimate of the value of conservatism in the diagnostic method employed was also made. For the most part, diagnosis was based upon clinical findings related to the site, amount and duration of blood loss. Treatment was generally conducted without knowledge of the morphology of the lesion. Intravenous pyelography was done on only 24 patients and then just prior to their discharge. Retrograde studies were employed only in 3 cases. In 5 cases which were selected for surgery because of signs of intraperitoneal rupture or recurrent bleeding with persistent nonfunction, pyelography was employed in follow-up studies.

Seventy-two patients were treated without surgery. There were no deaths or serious morbidity during their hospital stay. Five were explored and three nephrectomies were performed. Of 34 patients available for follow-up studies, only 1 was unwell. All had good renal function but 5 showed abnormalities on the pyelograms. One patient developed hypertension after the injury.

The authors feel that their findings support conservative management of renal trauma. Concerning the use of diagnostic procedures, they suggest that intravenous pyelography will give satisfactory information in the majority of cases when it is necessary to assess morphologic damage and, in addition, will demonstrate the presence and function of the opposite kidney. Routine retrograde urography might, in itself, increase the complications and it is believed to be undesirable in the majority of patients. —*Jack Reynolds, M.D.*

Platt, William R. Bilateral renal artery aneurysm: a cause of hypertension. *J. Urol.*, Jan., 1961, *85*, 24–30. (From: The Washington University School of Medicine, Department of Pathology and Missouri Baptist Hospital, Department of Pathology, St. Louis, Mo.)

In this article, the author presents a single case report of bilateral renal artery aneurysm in a twenty-two year old male patient with hypertension. Intravenous urograms showed that there were punctate calcifications in the region of the lower pole of the right kidney and the middle of the left kidney. The calyceal system was poorly defined; the renal parenchymal shadows were normal in size; and some of the minor calyces were moderately dilated, particularly in the region of the calculi. The roentgen examination of the chest showed some evidence of enlargement of the left heart compatible with the hypertension.

The past history of this patient indicated evidence of certain neurologic disturbances which suggested at least the possibility that he may have had an aneurysm of a cerebral vessel.

During his hospital stay, the patient developed severe pain in the left loin associated with signs of bleeding into the left kidney. At operation, a massive left renal hemorrhage was found and a left nephrectomy was done. The patient died in the operating room immediately after surgery.

Postmortem examination showed left ventricular hypertrophy. There was a large dissecting retroperitoneal hemorrhage throughout the abdominal wall on the left side extending to the suprapubic region. There was a large aneurysmal cavity in the lower pole of the left kidney. The left renal artery showed an aneurysm 6×4×2 cm. containing firmly adherent mural thrombi. The wall of this artery had focal areas of calcific and atheromatous change. The right kidney contained yellowish, friable calculi 3 to 6 mm. in diameter and the right renal artery showed an aneu-

rysm 2×3×5 cm. with a laminated reddish-gray mural thrombus.

Sections of the right and left kidney showed microscopically that there was thickening of the arteriolar walls and the lumina were slightly narrowed. The sections of the left kidney revealed an aneurysmal, thinned structure occupying the greater portion of the left lower pole of the kidney which compressed the adjacent pelvis and renal parenchyma. Microscopic study of the left and right renal artery showed that the intima was somewhat irregular and there was definite splitting of the elastic lamella with deposition of cholesterol and some destruction of the connective tissue fibers. The medial subintimal layers contained lipid-like material with associated hyaline-like plaques of fibrous tissue. The medial coat did not appear to be infiltrated by the fibrous elements such as are frequently associated with syphilitic lesions of the blood vessels. From these findings, it seems that the aneurysms are probably of arteriosclerotic origin with possibly a congenital weakness of the elastic fibers of the subintimal region and medial layer.

In the discussion of the etiology of hypertension, it is noted that this can occur with renal artery embolism, thrombosis, arteriosclerotic plaques, syphilitic arteritis, stenosis, and aneurysm formation. The hypertension in this particular individual could be explained by the narrowing of the orifices of the renal arteries which extended for a distance of 1 to 1.5 cm. It can be presumed, in this twenty-two year old male patient, that the bilateral renal artery aneurysms and the possible cerebral aneurysm were due to an underlying developmental defect upon which arteritis and arteriosclerotic phenomena were superimposed.—*George W. Chamberlin, M.D.*

BOERI, V., and RAMELLA, G. L'urografia endovenosa nei portatori di ipertrofia prostatica. (Intravenous urography in prostatic hypertrophy.) *Radiol. med.*, Aug., 1960, *46*, 708–728. (From: Istituto di Radiologia della Università di Genova, Italy.)

The authors review 210 cases of prostatic hypertrophy in which intravenous urography was done in the years 1957 and 1958. Their conclusions are as follows:

(1) Excretion urography in prostatism has a significance in the evaluation of renal function that may be compared to laboratory methods such as the blood urea, nitrogen, and phenolsulfonphthalein tests but with the advantage that it is not influenced by external factors or technical errors.

Although it does not replace clearance studies, it is useful and practical for the surgical evaluation of this type of patient.

(2) Intravenous urography permits an evaluation of the morphologic state of the entire urinary tract, of its function and of the reversibility of its lesions

(3) In the authors' opinion one of the principal advantages of excretion urography is that it allows one to decide on a therapeutic approach without the necessity of instrumentation, which is always dangerous in this type of patient: (a) good excretion, without evidence of alteration of the upper urinary tract, permits an unhurried surgical intervention determined by the general state of the patient; (b) poor excretion in the presence of nondilatation of the urinary tract indicates grave parenchymal insufficiency and requires a delay in surgery until such time as more precise studies may determine that the alterations are reversible; (c) poor excretion with dilatation of the upper urinary tract requires a period of bladder drainage before surgery, until the upper urinary tract returns to normal. Vesical drainage was obtained by a permanent catheter and only rarely was it necessary to perform suprapubic cystostomy; and (d) poor excretion associated with kinking of the juxtavesical portion of the ureter requires urgent prostatectomy, since it is impossible to improve renal function by means of vesical drainage. These cases are characterized by retention of the contrast medium in the upper urinary tract.—*Anthony A. Blasi, M.D.*

WATERHOUSE, KEITH. Voiding cystourethrography: a simple technique. *J. Urol.*, Jan., 1961, *85*, 103–104. (From: State University of New York, Downstate Medical Center, College of Medicine at Brooklyn, and the Department of Surgery, Division of Urology, Kings County Hospital, Brooklyn, N. Y.)

Previous descriptions of the technical procedures used for voiding cystourethrography have included the rather elaborate techniques of some Swedish authors who make simultaneous roentgen-ray exposures in the lateral and anteroposterior projection while the patient is voiding a suspension of barium sulfate. To do this, it is necessary to have a special swing-like cradle to support the patient and the technical difficulties associated with this procedure make it somewhat unsatisfactory for routine performance.

The author has reduced the technique to its simplest elements. He makes one roentgen-ray exposure with the patient voiding while lying in the oblique position on the roentgenographic table. This is the customary bicycle-riding position in which one thigh is flexed on the abdomen and the leg is flexed on the thigh while the opposite extremity is straight. A small, soft catheter is inserted and residual urine is measured. A solution of 30 per cent renografin is introduced into the bladder until the patient complains of distention. The patient is then placed in the oblique bicycle-riding position and one roentgenogram is made when he is actively voiding. In very young children, a similar technique may be carried out and the child may be given a bottle while waiting for micturition.

Voiding urethrograms are important for a number of reasons but particularly for evidence of dilatation

of the posterior urethra in association with posterior urethral valves.—*George W. Chamberlin, M.D.*

FEIST, JOHN H., and LASSER, ELLIOTT C. Pheochromocytoma with large cystic calcification and associated sphenoid ridge malformation. *Radiology*, Jan., 1961, *76*, 21–26. (From: Presbyterian Hospital, Pittsburgh 13, Pa.)

Pheochromocytoma is not a rare neoplasm, but calcification within this tumor is quite unusual. When calcification does occur, it is in the form of flecks and plaques, although there is 1 reported case of a right adrenal tumor containing a laminated calcific deposit resembling a gallstone. It is reported that 10 to 30 per cent of these tumors can be demonstrated by plain roentgenography, 20 to 50 per cent by urography, and approximately 50 per cent by retroperitoneal pneumography. The majority of the tumors demonstrated on plain roentgenograms are manifested by the presence of a soft tissue mass. It is known that 5 to 10 per cent of patients with pheochromocytoma also have neurofibromatosis or another variant of the neuroectodermal syndromes. The occurrence of such associated findings justifies roentgen examinations of the calvarium and a careful search for evidence of sphenoidal ridge hypoplasia in all cases of pheochromocytoma.

The authors report a large pheochromocytoma in a seventy-four year old white male. Calcification in the capsule of a central necrotic cyst facilitated the preoperative roentgen identification. Roentgenographic examination of the skull showed gross asymmetry of the calvarium with elevation of the left petrous pyramid, attenuation of the left sphenoid ridge markings, and asymmetry of the orbits. The surgically removed tumor measured 8×7×6 cm. The pathologic diagnosis was pheochromocytoma of the right adrenal gland, with cystic degeneration. The patient died one day following surgery. Necropsy confirmed the roentgenologically predicted deformities of the calvarium and the resultant enlargement of the left middle cranial fossa.—*A. W. Sommer, M.D.*

SKELETAL SYSTEM

YENTIS, IRVAN. Radiological aspects of myelomatosis. *Clin. radiol.*, Jan., 1961, *12*, 1–7. (From: Department of Radiology, King Edward VII Hospital, Windsor, England.)

There are three main varieties of myelomatosis: (1) lesions involving the bone marrow, including multiple myeloma and solitary myeloma; (2) extramedullary lesions, involving (a) the upper respiratory tract (sinuses, larynx, and nasopharynx), (b) the lymph nodes, and (c) other sites; and (3) plasma cell leukemia. The roentgenologic changes in bone, which are considered in this paper, are important because most of the lesions are located here and are roentgenologically significant. However, in considering the bone changes it must be remembered that the invaded skeleton may look completely normal to the radiologist.

The punched-out clear cut, rounded translucencies which are said to be typical of myelomatosis are seen best in the skull vault. The internal erosion of the cortex may produce a characteristic sharply scalloped appearance. Mandibular deposits are not uncommon. Soft tissue extensions are so frequent that their presence is a useful indication of disease in the ribs, where they form subpleural nodes and masses. Local rib expansion and pathologic fracture are commonly seen. Occasionally, plasma cell tumor may have a trabeculate appearance and, if assumedly single, it may simulate osteoclastoma. However, giant cell tumors tend to appear in young people, in the twenty to thirty year age group, whereas plasmacytoma is rare before the age of forty. A very common form of bone change is osteoporosis, seen best in the spine where the uniform trabecular absorption and cortical thinning are often associated with vertebral collapse.

Hyperparathyroidism, secondary carcinomatosis, Cushing's disease, osteomalacia and senile osteoporosis may have to be considered in the differential diagnosis. While it is generally held that "primary" myelomatous bone lesions are entirely osteolytic, new bone formation may occur: (a) as callus following fracture; (b) following treatment (radiation therapy or urethane); and (c) as a result of amyloid deposition. The author has recently seen three examples of bone spiculation in multiple myeloma.

Commenting on the subject of "solitary" myeloma of bone, the author feels that such apparently localized lesions, whether in bone or soft tissue, are part of a generalized disease. These tumors are "benign" only in the relative sense of being the least virulent manifestation of a malignant process, with plasma cell leukemia being the most virulent form. The possibility of frank dissemination is present in every patient with a "solitary" lesion.—*Samuel G. Henderson, M.D.*

BROMLEY, J. F. Myelomatosis and the radiotherapist. *Clin. radiol.*, Jan., 1961, *12*, 8–16. (From: Radiotherapy Department, The General Hospital, Birmingham 4, England.)

Over the past twenty years, views about the incidence, diagnosis and prognosis of myelomatosis, as regards the prolongation of useful life, have changed considerably. The author, in this paper, discusses 87 patients registered with the Department of Medical Statistics of the United Birmingham Hospitals in the years 1950 to 1956, from the standpoint of the value of roentgen therapy.

The age distribution shows a maximum incidence in the sixty to seventy year group with a preponderance of females. Backache in one form or another is

the most common symptom; other bone pains are second, and various chest symptoms third. Cough, bronchitis, pleurisy-like pains, various forms of hemorrhage, a painful lump on the breast bone or some form of abdominal disorder may be present. In the diagnosis, examination of the bone marrow is by far the most reliable single test. The apparently poor diagnostic quality of roentgenograms may be a true indication of the real state of the patient, who is often extremely ill, helpless and uncooperative.

In the present state of our knowledge there is no one successful method of treating this disease. The methods of treatment used in this series, with the number of patients so treated, include: (a) no treatment, 22; (b) surgery only, 4; (c) transfusion only, 5; (d) urethane, 17; (e) stilbamidine only, 2; (f) nitrogen mustard 2; (g) cortisone, 1; (h) "cytamen," 1; (i) triethylene melamine, 1; and (j) roentgen therapy (plus one P^{32}), 32.

A report of Tennant (1945) is cited concerning the case of a patient who lived five years from the onset of his symptoms without any treatment, and Bayrd and Heck (1947) described a patient operated on for paraplegia, due to an apparently localized myeloma of the first lumbar vertebra, who subsequently lived seven and one-half years without any other treatment. Results of this kind cannot help inspiring a healthy diffidence in claiming any one form of treatment as superior.

Of the 32 patients in this series treated by roentgen therapy, 5 are alive and well at four, five, six, eight and nine years, respectively. Three of the above have, to date, only solitary lesions, all proved histologically. Two patients who are alive and symptom free showed multiple tumors from the beginning. A woman aged forty-five has received over a period of four years courses of roentgen irradiation to 10 different areas. A man aged fifty-six years continues to lead a useful life eight years after initial roentgen therapy.

The author believes that roentgen therapy offers the most effective method of relieving pain and keeping the patient ambulatory. Dosage is quite variable because the disease is one which particularly demands individual treatment planning. The solitary, or apparently solitary, tumor justifies a more radical order of dosage, even in the presence of a positive bone marrow examination. In the presence of initial severe anemia and weakness in a patient of the older age group, it is inadvisable to use a high dosage.

The two separate aims of the radiotherapist are quite clear. In the case of the solitary tumor it is to obtain healing and to prevent the disease from becoming generalized; when the disease is obviously generalized, the effort is directed to keeping the patient free from pain and encouraging him to leave his bed. The author has found that tumor doses of the order of 1,000 r over ten to fourteen days to individual lesions in bone can achieve this result as effectively as larger doses.

In this group of 87 patients the respective survival rates at two years are 4.5 per cent for no treatment, 5.9 per cent for urethane, and 58 per cent for roentgen therapy. The corresponding figures for three years are 4.5 per cent for no treatment, zero for urethane, and 38.7 per cent for roentgen therapy.—*Samuel G. Henderson, M.D.*

LEONARD, B. J. The pathology of myelomatosis. *Clin. radiol.*, Jan., 1961, *12*, 20–25. (From: Pathology Department, St. Mary's Hospital, London W.2, England.)

The finding of plasma cells in the bone marrow is an essential step in the diagnosis of myelomatosis. Other diseases which produce an increase in the plasma cells in the bone marrow include Hodgkin's disease, systemic lupus erythematosus, rheumatoid arthritis and carcinoma.

The normal plasma cell has an eccentric nucleus, a characteristic arrangement of the nuclear chromatin in small blocks and a deep basophilia of the cytoplasm due to a high concentration of ribonucleic acid. Myeloma cells are usually larger than normal plasma cells; they vary in size and have a less basophilic cytoplasm; the nucleus occupies a greater proportion of the cell; the cells are often multinucleated; and the nucleoli are large and distinct.

Myelomatosis is a neoplastic disease that predominantly involves those parts of the skeleton which contain red marrow in the adult. The skull, the vertebrae, the ribs, the sternum, the pelvis and the upper ends of the femora and humeri are the sites maximally involved.

Reticulosarcoma, Hodgkin's disease and chronic lymphatic leukemia have some features in common with myelomatosis. In multifocal reticulosarcoma destructive bone lesions are common. The absence of bone lesions in acute myeloid leukemia and in all forms of acute leukemia in the adult can probably be explained when it is remembered that the course of the disease is shorter than in children because drug induced remissions are uncommon.

It would appear that destructive bone lesions are not confined to myelomatosis, but are seen in an ever widening spectrum of blood diseases. Postmortem studies made in 38 of the 48 cases of myelomatosis show that the pathologic findings can be divided into a leukemic and a sarcomatous group. In 30 cases there was a diffuse plasma cell infiltration of the bone marrow, the spleen, the liver and the lymph nodes. The plasma cell infiltration in the liver was accentuated around the portal tracts similar to the distribution in chronic lymphatic leukemia. The three examples of plasma cell leukemia showed this pattern of infiltration. The findings support the concept that myelomatosis is a plasma cell leukemia with a prolonged aleukemic phase. The smaller group of 8 patients showed nodular infiltrations of the liver and spleen. The deposits were often large and very like those seen in reticulosarcoma.

Approximately 10 to 20 per cent of patients with myelomatosis develop paramyloidosis. Paramyloid is closely related to amyloid but has different staining properties, and its distribution in various organs is different from that seen in amyloidosis. Paramyloid in myelomatosis is seen mainly in the tongue, the gastrointestinal tract, the heart, the joints, the skin and the peripheral nerves. Amyloidosis developed in 6 of 48 patients with myelomatosis, in 3 of 40 with Hodgkin's disease, and in 2 of 30 with chronic lymphatic leukemia.

The association of amyloidosis with chronic lymphatic leukemia introduces the problem of what part the neoplastic lymphocyte plays in the formation of this abnormal protein. Can the lymphocyte as well as the plasma cell produce abnormal proteins? Myelomatosis, reticulosarcoma, Hodgkin's disease and chronic lymphatic leukemia are all closely related neoplastic diseases derived from the reticulum cell. They are all invariably fatal and, at least terminally, behave as malignant tumors. All these conditions give rise to destructive bone lesions and, with the exception of reticulosarcoma, they are associated with the formation of amyloid.—*Samuel G. Henderson, M.D.*

RADIOISOTOPES

GOTTSCHALK, RAYMOND G. Radioactive sulphur in chondrosarcomata; distribution, concentration, and effects of S^{35} after intravenous administration to patients with chondrosarcoma. *J. Bone & Joint Surg.*, Oct., 1960, *42A*, 1239–1257. (From: Department of Pathology, George Washington University School of Medicine, Washington, D. C.)

Several recent papers have called attention to the fact that radioactive sulfur (S^{35}) may be incorporated in chondroitin sulfate within chondrosarcomas. The author gives the results of S^{35} tissue studies in 5 patients known to have chondrosarcomas and in 1 who had an undifferentiated sarcoma arising in muscle.

In distribution studies it is interesting to note that initial uptake of S^{35} is greater in hematopoietic bone marrow than it is in cartilage, but this concentration is transient, whereas the rate of disappearance of S^{35} from cartilage, and cartilage-derived tissues, seems relatively slow. Distribution figures also show that one chondrosarcoma, described as poorly differentiated, retained a fairly uniform concentration of the isotope over a period of eleven days between administration and autopsy.

This suggests that there may be some role for radioactive sulfur in the management of malignant tumors of cartilaginous origin, as other authors have suggested. A number of interesting radioautographs are presented.—*Frederick J. Bonte, N.D.*

MISCELLANEOUS

BICKEL, WILLIAM H., CHILDS, DONALD S., and PORRETTA, CHARLES M. Postirradiation fractures of the femoral neck; emphasis on the results of treatment. *J.A.M.A.*, Jan 21, 1961, *175*, 204–212. (From: The Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Postirradiation fractures of the femoral neck were studied in 19 patients. In 5 patients the fractures were bilateral so that the total experience included 24 fractures. The lesions for which pelvic irradiation had been given included carcinoma of the rectum and lymphosarcoma but the predominant lesion was carcinoma of the female genital tract. The interval between the initial dose of roentgen rays and the appearance of a pathologic fracture varied from five months to twelve years, the majority of fractures occurring within five years.

There was considerable variation in the amount of radiation administered and the time during which it was given. The dose to the neck of the femur ranged from 1,200 to 3,700 r. All patients were treated with 130 to 250 kv. equipment. Since the energy absorbed by bone at these energy levels is approximately twice that of soft tissue, the authors estimate that the dosage range to the femoral necks was approximately 2,400 to 7,400 rads.

The first sign of impending fracture frequently was pain, which in many cases was not sufficiently severe to stop activity. Gradual onset of difficulty was the rule, although in several cases the fractures were rather acute, following minimal stress. There was no history of severe trauma in any patient. Roentgenograms taken at the onset of symptoms often failed to show a fracture. In some cases the negative roentgen findings led the physician to suspect early metastasis to the pelvic or hip region.

The treatment used in the 24 fractures varied from none to reconstructive operations. Thirteen fractures had nonoperative care, and union occurred in all 13. It appears that if a program of crutch walking is instituted, union occurs in about the normal period for a fractured neck of the femur (twelve weeks) even though the fracture is recognized late and even though there is some varus deformity.

Seven fractures were fixed with either pins or triflange nails, and union occurred in all. Internal fixation was used when the fracture did not appear stable, as shown by progressive slipping or separation of the fragments. One patient with a nonunion had walked with and without crutches for seventeen months. A Whitman reconstruction with a cup was performed but the result was poor because of an infection. Prostheses were inserted in two instances of nonunion. In these cases the fractures had been present for five and six months, respectively, and the shaft was markedly displaced. Although the results were good the authors question whether a union

might have occurred if the fractures had been treated with simple fixation or a fibular bone graft. No instances of avascular necrosis of the femoral head were seen.

A postirradiation fracture may be difficult to differentiate from a pathologic fracture through a tumor. In the roentgenograms, an early postirradiation fracture first resembles a pseudofracture and later a true fracture. There is rarely any erosion such as one would expect in a metastatic lesion. In the course of this study the authors saw 28 pathologic fractures of the femoral neck due to malignancy but in no instance was the primary lesion in the genital tract.

The authors state that moderate doses of roentgen rays cause an early hyperemia. This promotes the osteoporosis seen in these hips before fracture occurs. There is some tissue death and fragmentation of the trabecular pattern with loss of structural strength and a predisposition to stress fractures. Microscopically, the vascular supply to the femoral head does not appear to be greatly damaged.

It is concluded that the majority of postirradiation fractures of the femoral neck will heal rapidly and well with simple nonweight bearing treatment. Surgical procedures may be necessary if the fracture is old or the position is poor. Avascular necrosis is rare.—*J. L. Williams, M.D.*

HALLERBACH, H., and RENNER, K. Struma maligna als Strahlenschädigung? Zugleich ein kasuistischer Beitrag zur Statistik der Struma maligna. (Struma maligna a radiation damage? Also case report contribution to the statistics of struma maligna.) *Strahlentherapie*, Sept., 1960, *113*, 110–120. (From: Strahlenklinik Professor Dr.h.c. R. Janker, Bonn, Germany.)

Statistical study of 37 patients admitted with a diagnosis of struma maligna showed that 9 of 18 patients observed for five years were "cured," and that, of 11 patients with histologically proved malignancy, 7 had five year "cures."

Although 4 of these 37 adult patients had received radiation therapy (for a gynecologic carcinoma, an arthrosis of the knee, a goiter, and symptoms probably due to thyroid carcinoma), in none did a cause and effect relationship seem plausible. However, reports indicate that one-third of struma maligna in children may stem from antecedent irradiation, so the authors recommend that irradiation (including administration of I^{131}) of the cervical region in children be undertaken only for pressing indications.—*Henry G. Moehring, M.D.*

KÖHLER, J. Herpes zoster nach Röntgenbestrahlung. (Herpes zoster following roentgen irradiation.) *Strahlentherapie*, Aug., 1960, *112*, 587–594. (From: Röntgen- und Strahlenabteilung der Krankenanstalten des Kreises Aue/Sa., Germany.)

A review of the author's 12 patients who developed herpes zoster after radiation therapy and of patients reported by others (a total of 97) failed to convince him of a cause and effect relationship between irradiation and herpes zoster. He states, deliberately and cautiously, that many factors *may* combine to activate a "latent" zoster virus and that perhaps roentgen irradiation is one of these factors.—*Henry G. Moehring, M.D.*

BÖRNER, W., MOLL, E., SCHNEIDER, P., and STUCKE, K. Zur Problematik der Thorotrastschäden; klinische und radiologische Untersuchungen zum Verhalten von Thorium und seiner Zerfallsprodukte im Organismus; I. Mitteilung. (The problem of thorotrast injury; clinical and radiologic investigations on the content of thorium and its disintegration products in the body; first communication.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1960, *93*, 287–297. (Address: K. Stucke, Chirurg. Univ.-Klinik, Würzburg, Germany.)

The report is based on examinations and observations of 8 patients who received intra-arterial thorotrast injections for arteriographic purposes fifteen to twenty-three years previously. In all of them, extensive laboratory studies of the blood, urine, liver function, and electrophoresis were performed. The results in none were definitely abnormal. In 2 patients specimens of the liver and spleen were obtained by surgery. Five of the 8 patients were asymptomatic. There was no apparent genetic damage. Of the 6 married patients, 1 was pregnant and 4 had healthy children.

For evaluation of concentration and localization of the thorotrast deposits, the roentgenographic examinations for search of abnormal densities and surface scintillation surveys were used. The investigation was done topographically. The maximum count representing 2 to 5 times of the O-effect was obtained over the liver and spleen. This is explainable by the storage of the thorotrast in the liver, spleen, and lymph nodes due to the known avidity of the reticulum cell system for thorotrast. A definite connection between the thorotrast deposits and the original site of injection could not be clearly established. In 1 patient, however, who had had injection into the left carotid artery, the liver and spleen showed low counts. This was not observed in other patients in whom carotid and vertebral angiographies were performed. The amount of injected material is not specified.

In 2 patients, the spleen was removed therapeutically because of high storage of thorotrast seen in roentgenograms and on scintillation counts. Histo-

logic examinations of liver biopsy specimens presented a picture of small, nodular, perilobular liver cirrhosis with deposition of thorium around the central vein and main distribution around the periportal fields. Fatty degeneration was marked but large segments of the liver parenchyma did not appear altered. The regenerative repair activity was not definitely present. The changes in the spleen consisted of marked deposition of thorium in the trabeculae with fibrosis.

The authors feel that removal of the spleen as the main thorium storage organ is indicated.—*Nathan Kriss, M.D.*

Krückemeyer, K., Lessmann, H. D., and Pudwitz, K. R. Nierenkarzinom als Thorotrastschaden. (Kidney carcinoma as the result of thorotrast injury.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1960, *93*, 313–321. (Address: K. Krückemeyer, Städt. Krankenh., Karl [Marx Str. 142a, Berlin-Neukölln, Germany.)

Carcinoma of the left kidney is reported in a forty-five year old patient who had retrograde pyelography with thorotrast nineteen years previously.

The initial pyelograms showed mild hydronephrosis due to a suspected stone on the left and minimal changes on the right. The late studies demonstrated accumulation of opaque thorotrast in the kidney parenchyma on both sides with preserved function on intravenous pyelograms on the right and complete lack of excretion on the left. A deformity of the lower pole on the left was present, indicative of a neoplasm. The scintillation counts over both kidneys were increased 17 to 23 times of the background.

On operation an adenocarcinoma was found in the lower pole of the left kidney which was markedly atrophic and hydronephrotic. The connection between thorium deposits and the carcinoma was assumed on the basis of distribution of the agent.

Generally, in animal experiments the thorotrast stimulates the connective tissue rather than the epithelium to produce malignant lesions resulting in sarcomatous growth.—*Nathan Kriss, M.D.*

Dihlmann, W. Zur Morphologie, Theorie und Problematik der Strahlenspätschäden im Zentralnervensystem. (The morphology, theory and problems of late radiation damage to the central nervous system.) *Strahlentherapie*, Aug., 1960, *112*, 567–586. (From: Neuropathologisches Institut der Universität, Tübingen, Germany.)

Late radiation damage in the central nervous system differs from local radiation damage eleswhere only in that, because of the unique character of the brain, the clinical course is much more malignant.

From 5,300–6,000 r tissue dose (in daily increments of 200 r) approaches the tolerance limit in the central nervous system. This tolerance varies. Associated factors may decrease the tolerance, *e.g.*, cerebral edema, systemic illnesses or drugs which alter vascular permeability or circulatory efficiency. On the other hand, increased tolerance may occur, as evidenced by reports of no late cerebral damage after 9,000 r tissue dose; no late damage four years after a tissue dose of 15,000 r; and late cerebral damage in only 6 of 71 patients who received 6,000 r or more cerebral dose.—*Henry G. Moehring, M.D.*

Hennig, K. Unerwünschte Radiojod-Inkorporationen und ihre Verhütung. (Undesired uptake of radio-iodine and its prevention.) *Strahlentherapie*, July, 1960, *112*, 462–466. (From: Strahlenklinik der Medizinischen Akademie, Dresden, Germany.)

Weekly direct contact measurements of radioactivity over the thyroid gland of 3 isotope technicians showed up to 1,849 counts per minute. Similar measurements in 6 euthyroid men and 4 euthyroid women 24 hours after the oral administration of 0.12 μc of radioiodine averaged 514 counts per minute. Since the average 24 hour uptake value is 55 per cent, 935 counts per minute indicate 100 per cent uptake of the 0.12 μc; and 0.12 μc maintained in the thyroid gland yields 0.3 rem, the maximum "permissible dose." Five of the thirteen weekly counts obtained in the isotope worker charged with the measurement and administration of radioiodine therapeutic doses exceeded this "permissible" count.

Control measurements excluded ingestion *via* contaminated hands. Radioiodine was being used in ion form as a sodium iodide solution (pH 5.4–7.5). Fifteen patients referred for radioiodine diagnostic study without previous exposure to radioactive isotopes sat for a short time (15–45 minutes) at a definite distance (35 and 85 cm.) from a beaker containing a therapeutic dose (16.4–25 mc) of radioiodine. In 24 hours their thyroid glands showed direct contact counts of from 35 to 8,359 per minute. Inhalation of vaporized radioiodine seemed to be the only explanation for this uptake.

The addition of sodium bicarbonate in order to alkalinize the sodium radioiodide solution (to a pH of 7.8) suppressed the uptake by inhalation to 0–54 counts per minute. Cooling the sodium radioiodide solution suppressed the uptake only when the solution was alkaline or neutral.

The author explains the inhalation not on the basis of radioiodine being incorporated in vaporized water, but by an oxidation of the iodide ion to elementary iodine; oxygen and the dissolved carbonic acid play an essential role in this process.

Alkalinization of the sodium radioiodide solution with sodium bicarbonate prevents vaporization (and inhalation) of the radioiodine.—*Henry G. Moehring, M.D*

CAROTID ARTERIAL INSUFFICIENCY— *SURGICAL CORRECTION*

Radiograph on Kodak Blue Brand X-ray Film; surgery photographed on Kodak Ektachrome Film

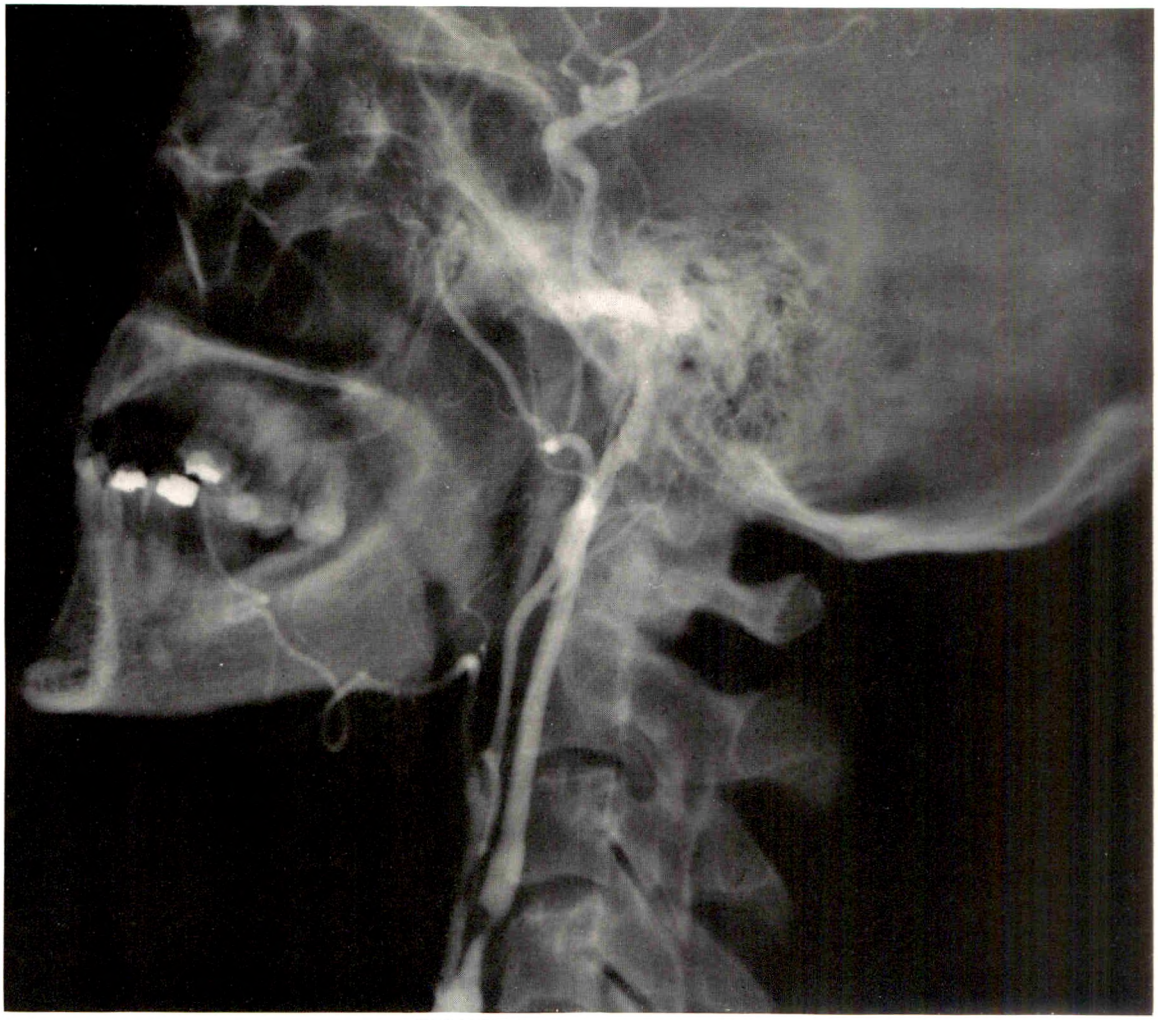


FIGURE 1.

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Figure 1: Preoperative carotid arteriogram shows carotid arteries with sclerotic narrowing of the internal carotid artery at its origin. **Figure 2:** Incision along anterior

CAROTID ARTERIAL INSUFFICIENCY— *SURGICAL CORRECTION* (cont'd)

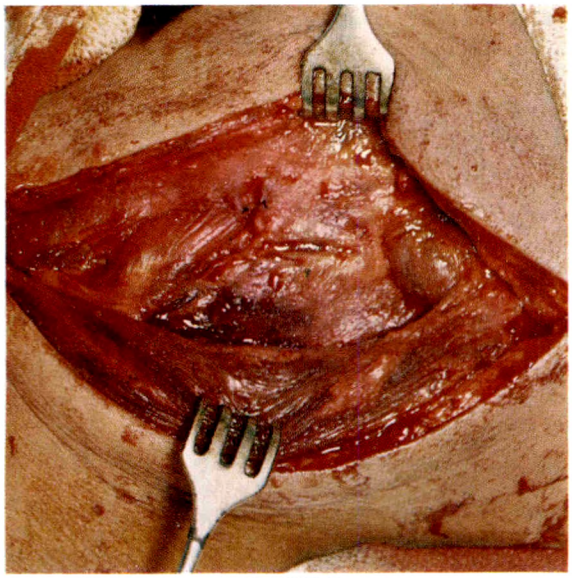


FIGURE 3.

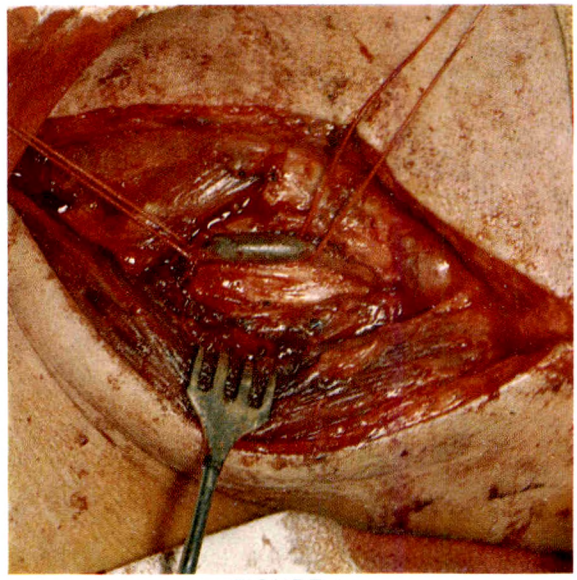


FIGURE 4.

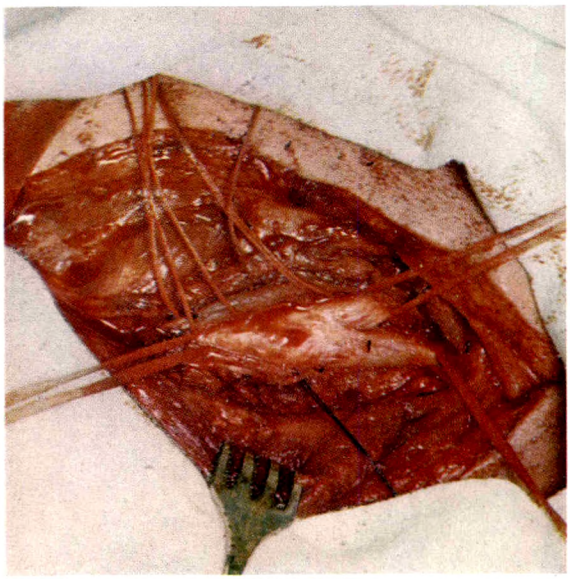


FIGURE 5.

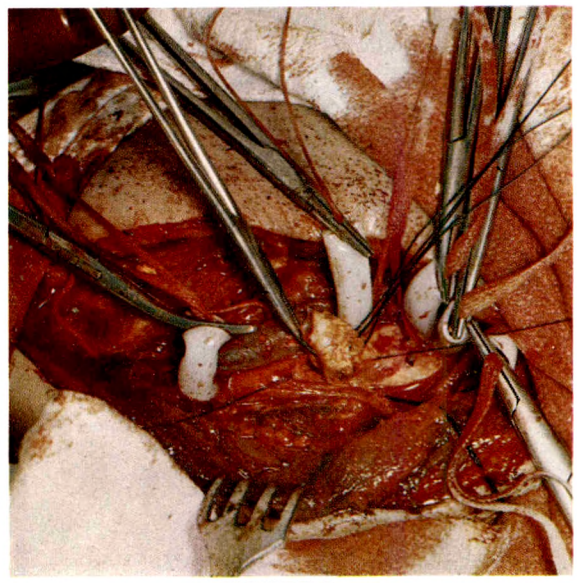


FIGURE 6.

Figure 3: Retraction of sternocleidomastoid muscle exposes deep cervical fascia. (Parotid gland now visible at upper pole of incision.) **Figure 4:** Tapes in place about internal jugular vein. **Figure 5:** Tapes around the carotid arteries. **Figure 6:** Internal carotid artery open and arteriosclerotic "core" being removed. *Note how effectively color establishes tissue differentiations.*

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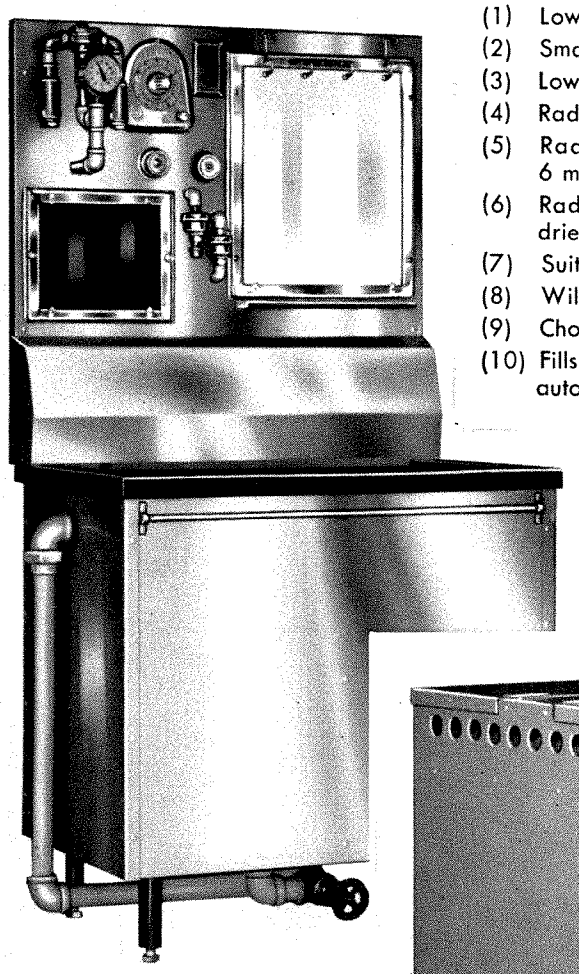
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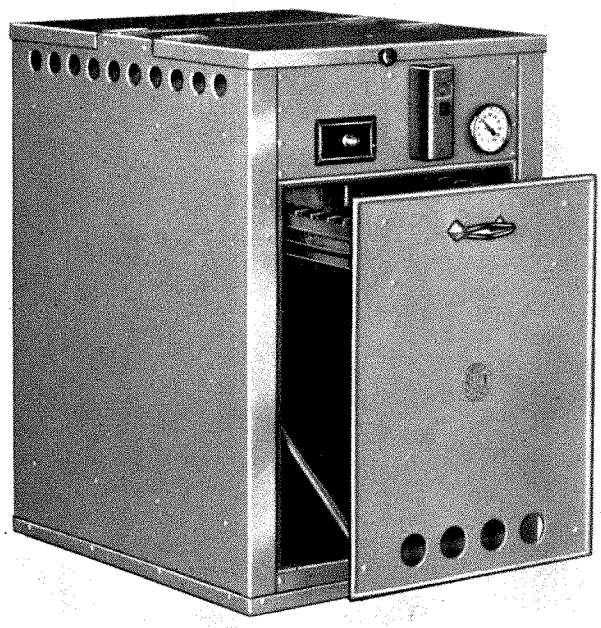


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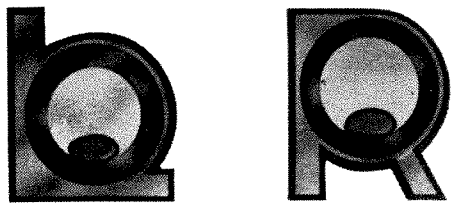
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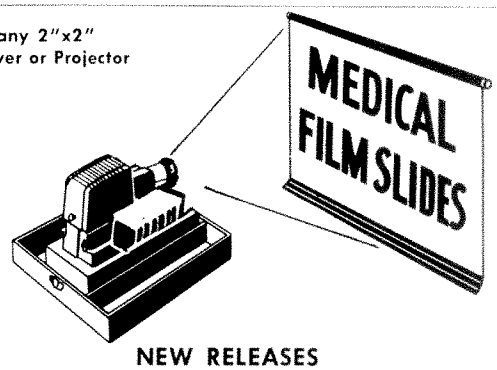
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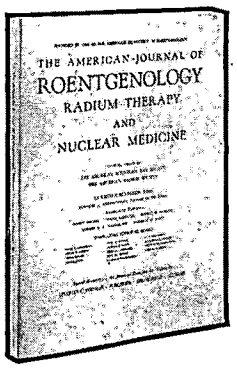
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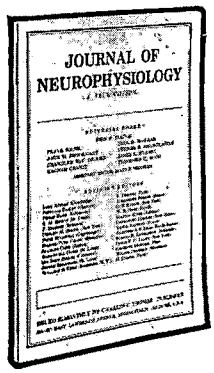
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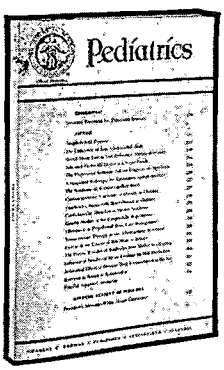
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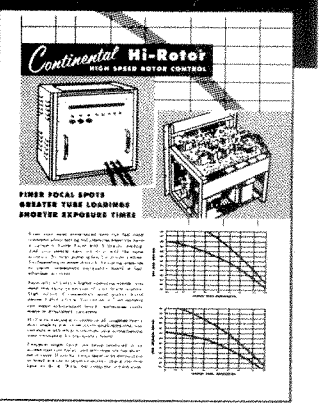
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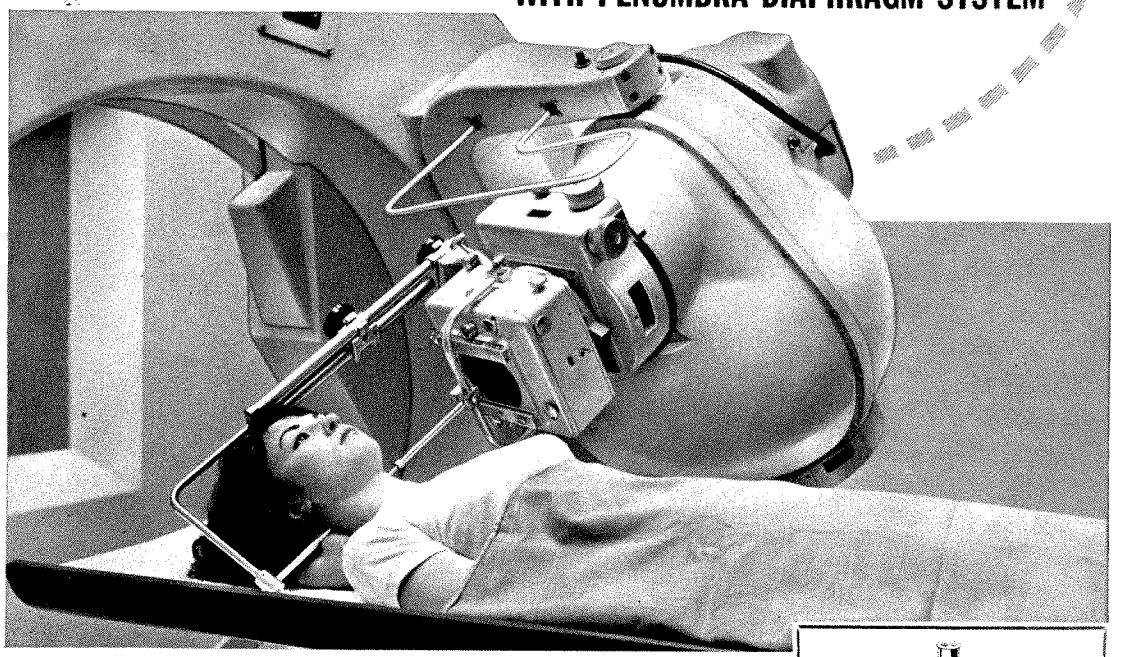
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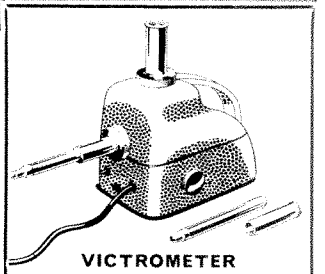


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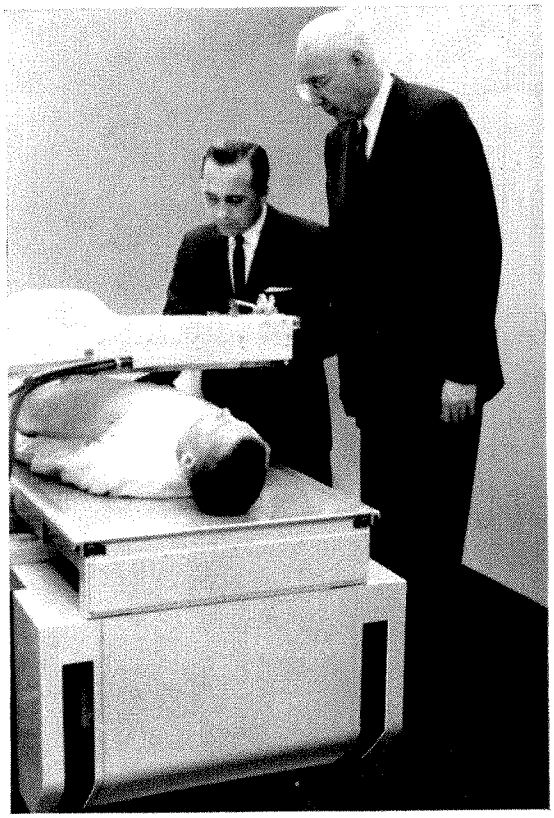
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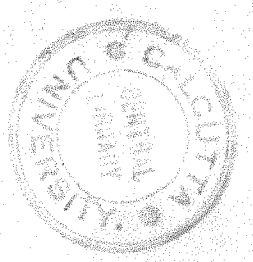
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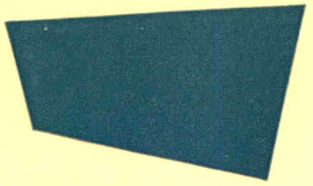
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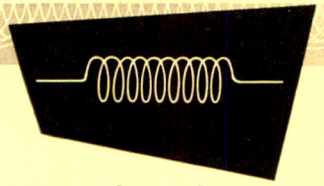
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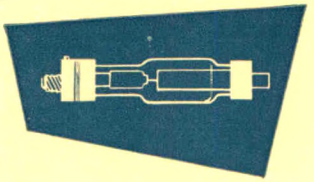
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Entered as Second-class at Springfield, Illinois under the
Act of March 3, 1879. Printed in U.S.A.

Second-class postage paid at Springfield, Illinois
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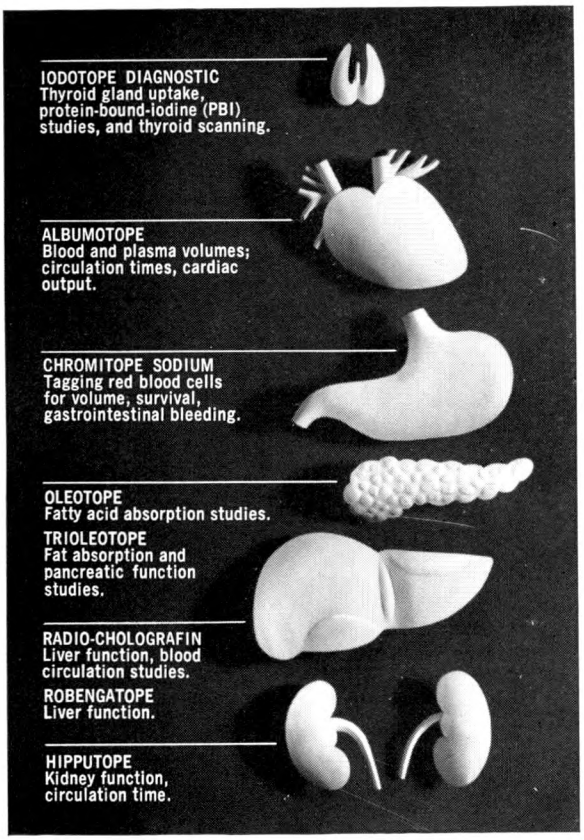
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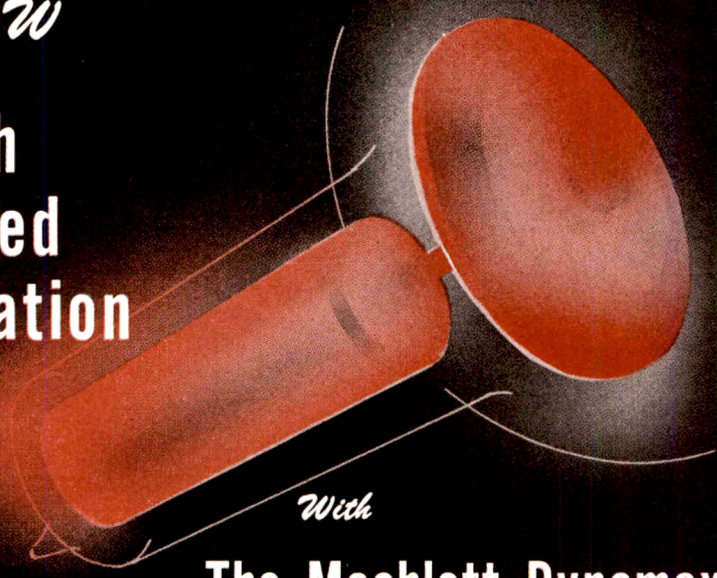
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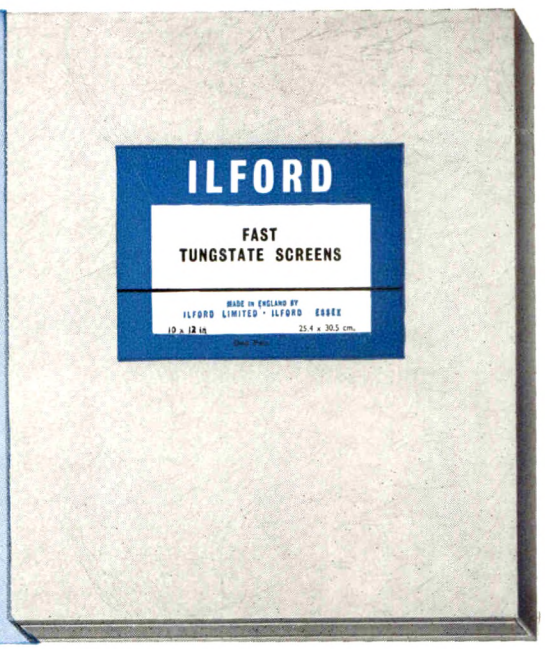
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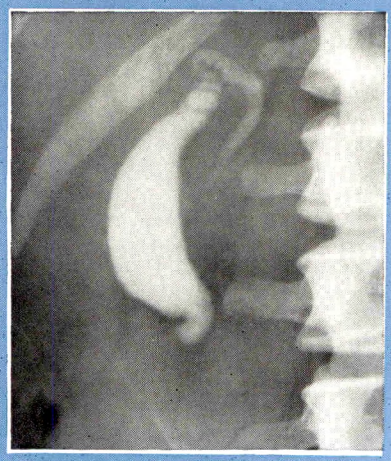
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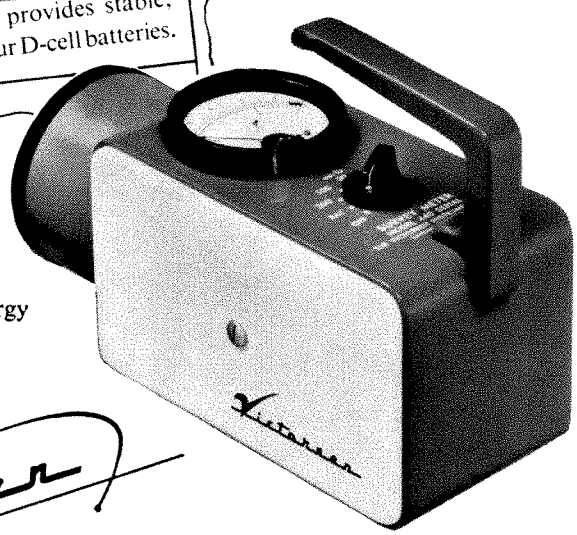
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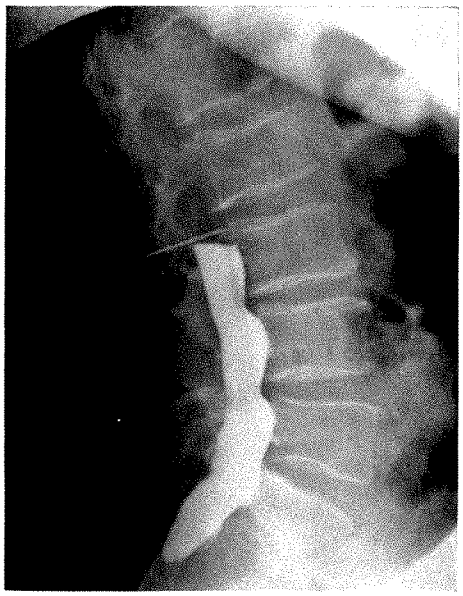
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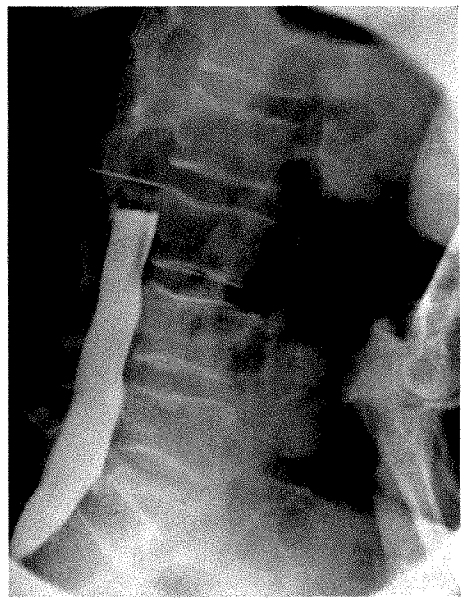
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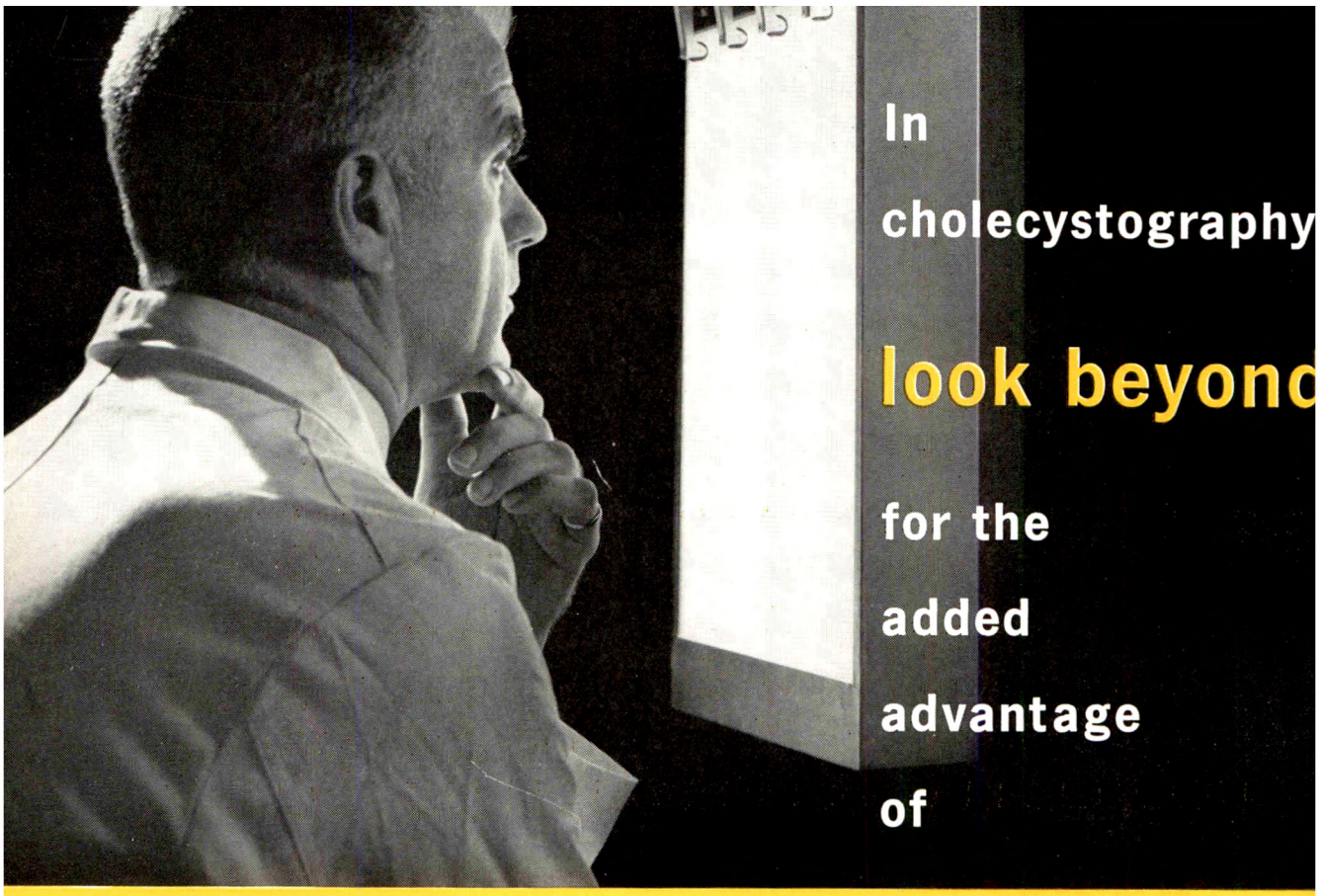
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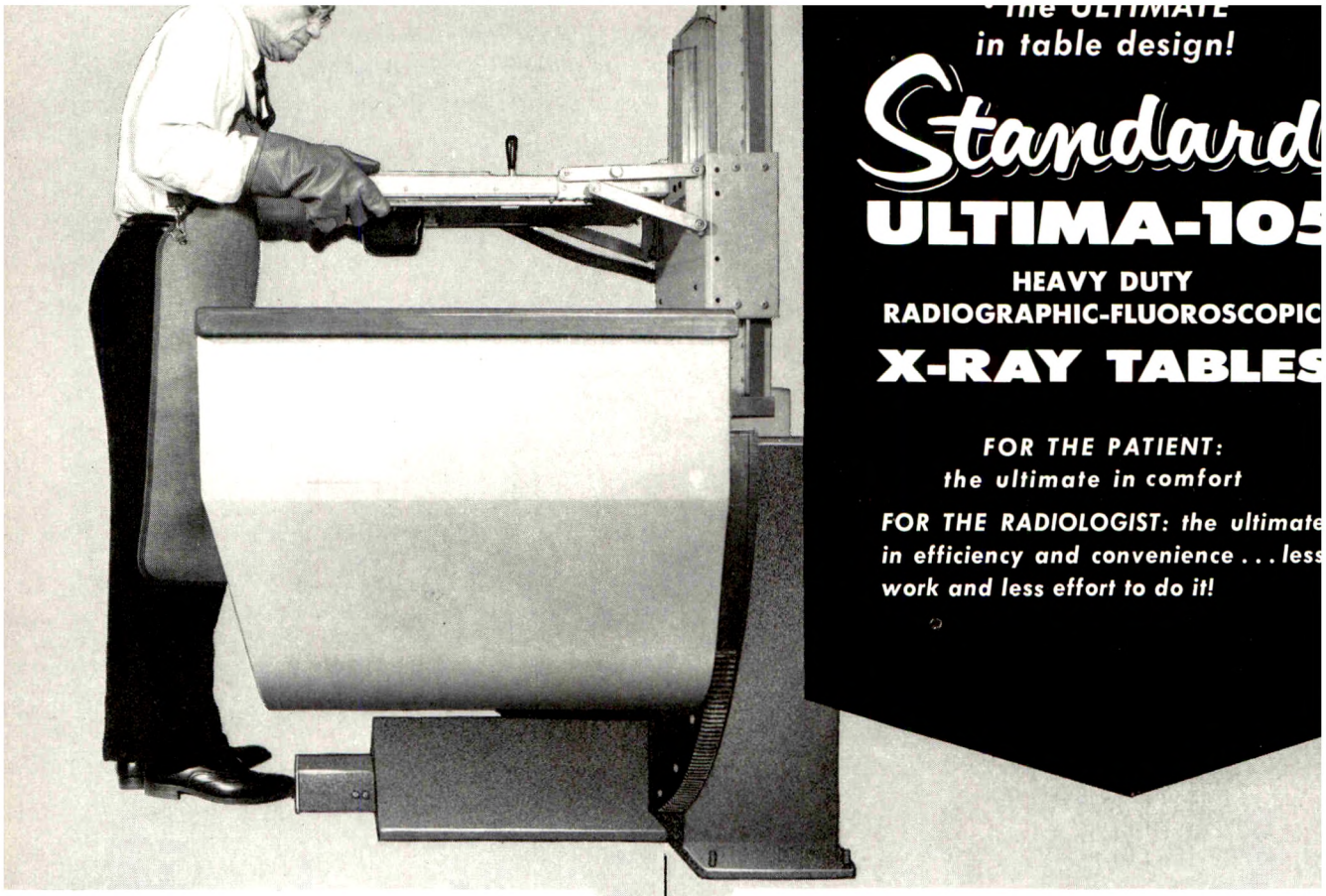
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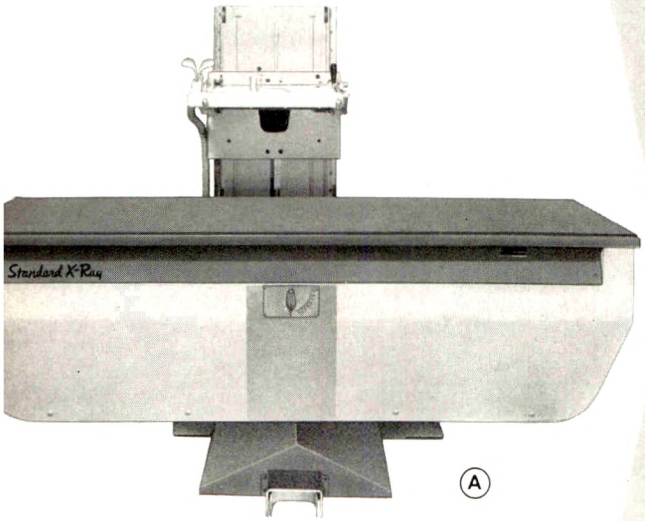
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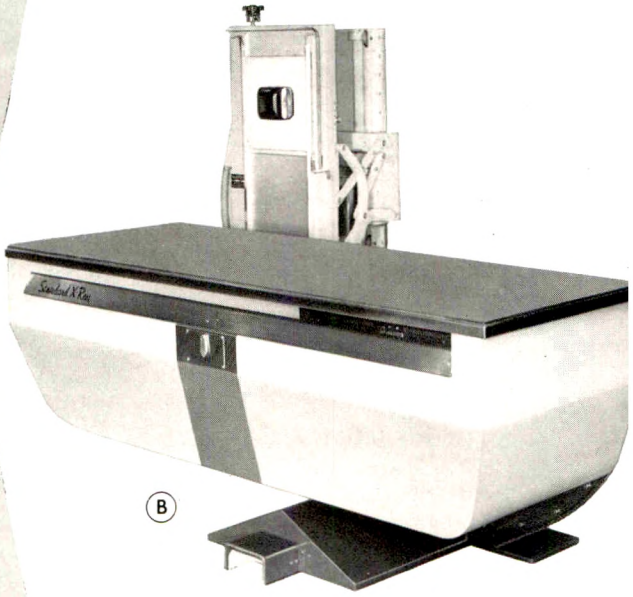
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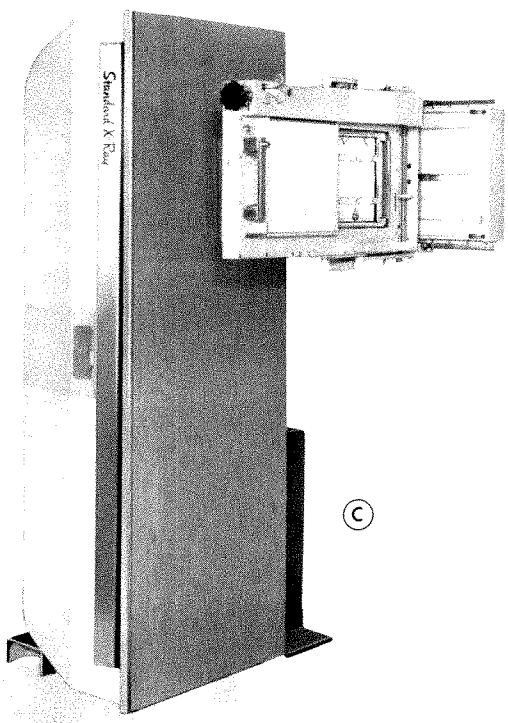
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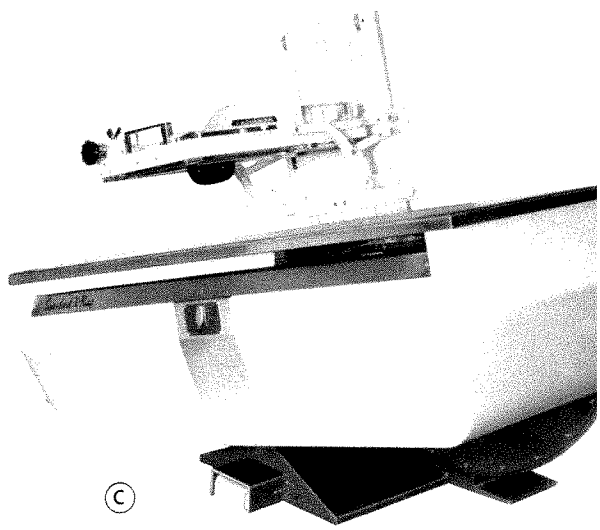
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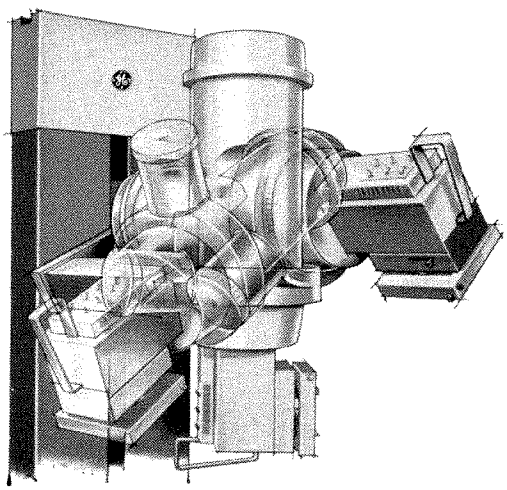
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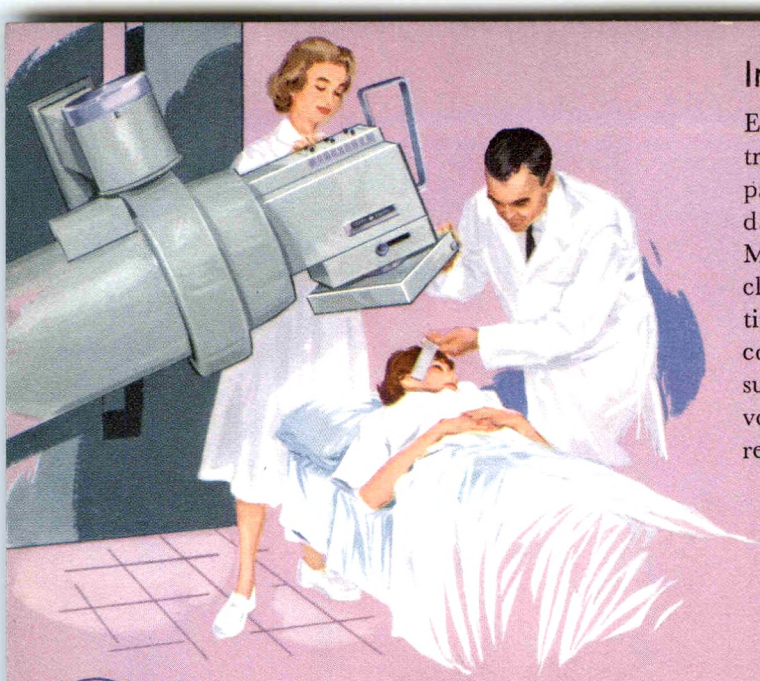
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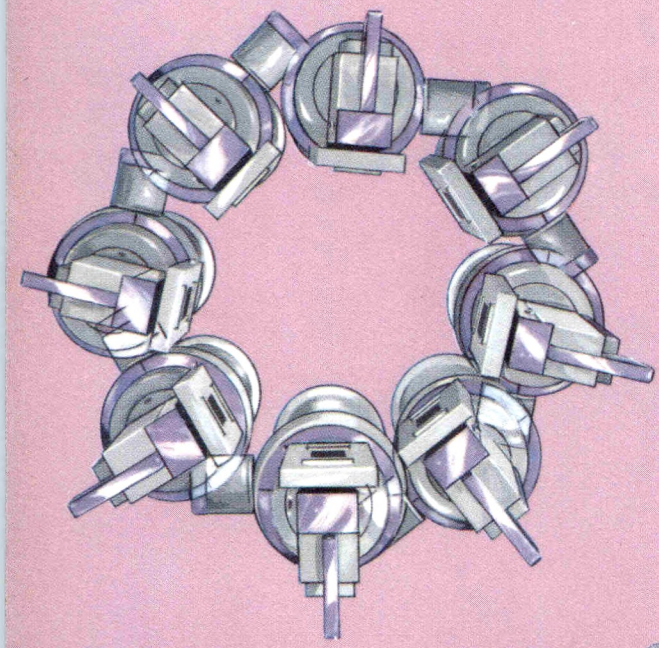


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clip minutes from preparation-for-treatn
time. High *r*-output speeds actual treatn
constant dosage assured by electron
supply, stabilizing both tube current
voltage. Operator's time is conserved
reliable remote controls described belo

MAXITRON® 300

X-RAY THERAPY UNIT



All-around operating flexibilit

Whether you mean "handling ease" v
say flexibility—or are thinking of freedo
tion without limitation—Maxitron 30
Up, down, in, out, across and around
compact, gas-insulated tubehead
smoothly and easily to guidance from y
...locks "on target" with the flip of a swi
ease of adjustment greatly facilitates i
intracavity, multiport or rotational app

Remote technic selection

Dial the exact dose rate and half-value layer you want, any of *ten* directly from control console (half-value layers range from 0.25-mm Al to 4-mm Cu). Correct filter spins into position, kvp and ma are matched—all automatically. Vernier adjustment of kvp and ma, registered on direct-reading meters . . . built-in beam monitor to check dose rate . . . precision, integrating-type, electric treatment timer . . . many other conveniences you'll appreciate in this compact control.



Check all MAXITRON 300 advantages for RANGE OUTPUT CONVENIENCE

SPECIAL NEWS FOR OWNERS OF GENERAL ELECTRIC X-RAY EQUIPMENT



NOW YOU CAN SAVE ON SERVICE WITH YEAR-ROUND PLANNED MAINTENANCE!

G-E x-ray equipment owners everywhere now can share in a unique new program called *Planned Maintenance Service*. It tackles x-ray service problems from two angles: First, through regularly scheduled "tune-ups" of equipment . . . second, by providing emergency service any time during business hours, free of further labor charges.

Curb repair costs. Pay only the monthly rate and, thereafter, you're free of service-labor charges no matter how serious the problem or how frequently you request help!

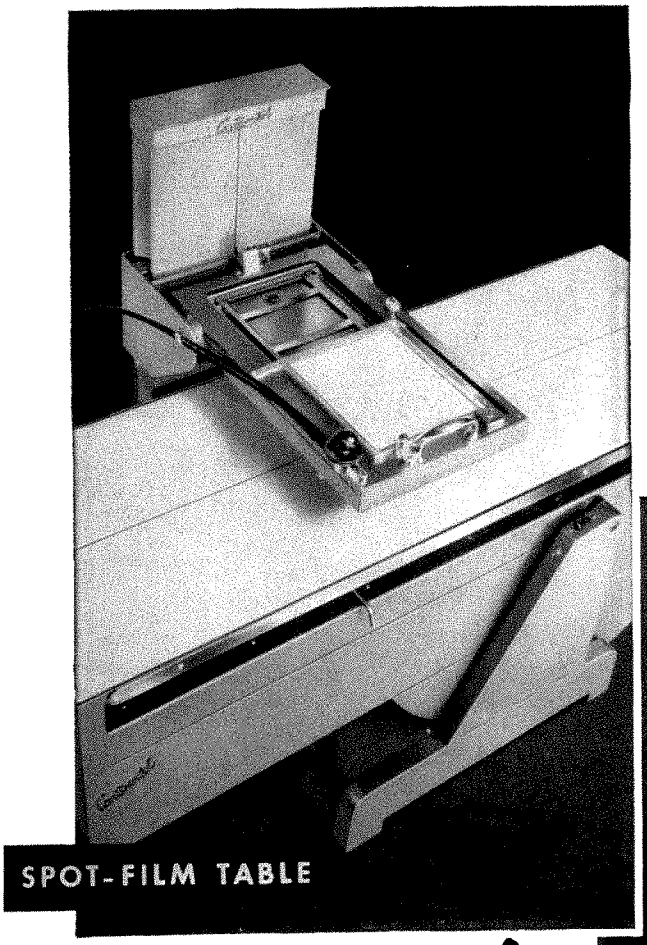
Extend equipment life. By detecting trouble early and correcting it, apparatus life is prolonged . . . premature deterioration forestalled. Performance stays at unusual levels of efficiency.

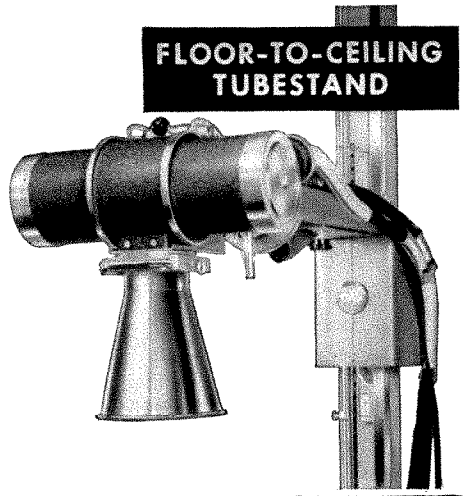
Easier budgeting. This bonus advantage stems from never being in the dark about future labor costs for x-ray repairs. Makes it easy to establish a figure for your annual budget.

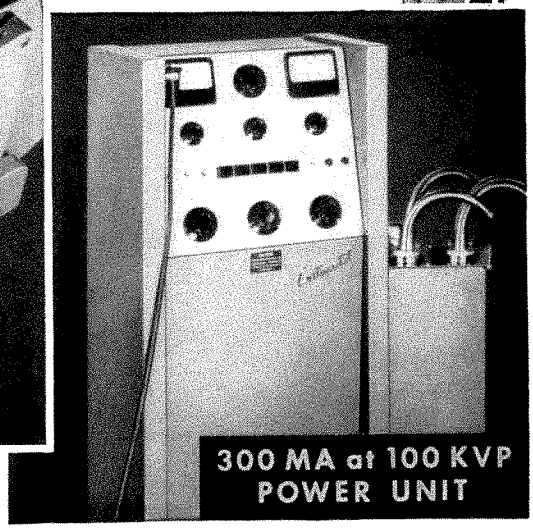
Full details on *Planned Maintenance Service* can be obtained from your G-E x-ray representative. Or write to X-Ray Department, General Electric Company, Milwaukee 1, Wisconsin, requesting Pub. A-82.

Progress Is Our Most Important Product

GENERAL ELECTRIC





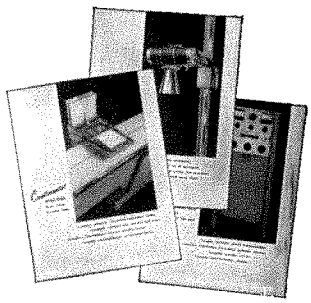


Continental SP-300

A complete X-ray installation of unprecedented reliability—surpassing anything in its power range—***but priced considerably lower.*** CONTINENTAL has used the packaged assembly concept to reduce the cost of producing this elaborate X-ray equipment.

COMPLETE

300 MA at 100 KVP automatic power unit
Motor-drive, spot-film table
Floor-to-ceiling, 360° tubestand
2 rotating anode X-ray tubes



Write for information on this new equipment; no obligation.

CONTINENTAL X-RAY CORPORATION

1536 Clybourn Ave.
Chicago 10, Illinois

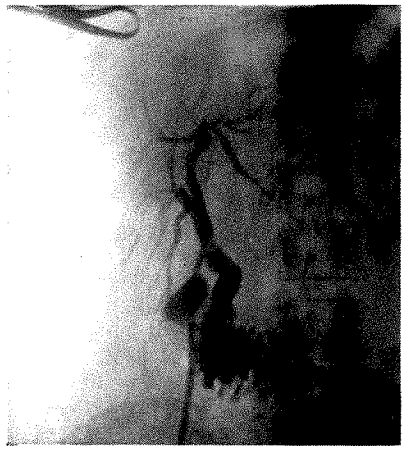
68 Nassau St.
New York 38, N. Y.

MD/RR
minimal dosage/rapid radiography

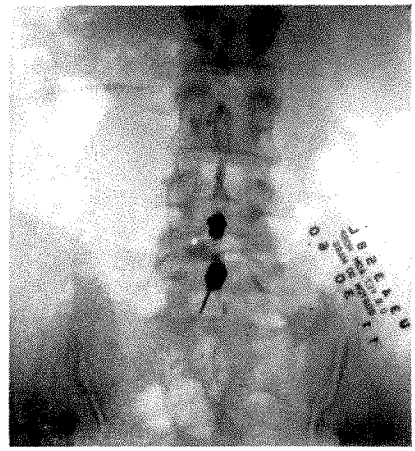
lightning fast, radiation-sparing x-ray
for procedures like these where SECONDS count



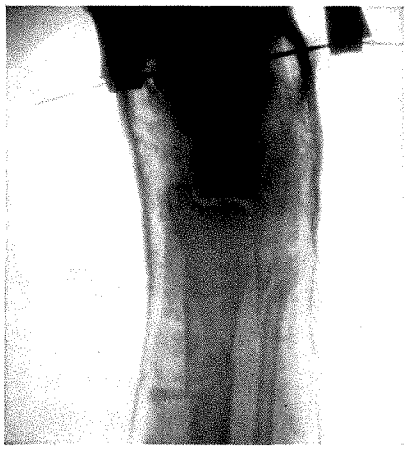
STEREOTAXIS



CHOLANGIOGRAM



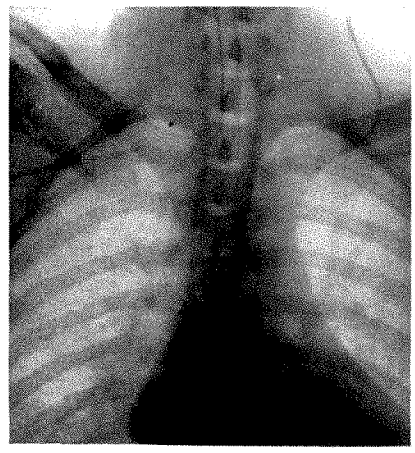
DISCOGRAM



TIBIA/FIBULA (WET CAST)



HIP PINNING



CATHETER LOCATION

POLAROID 3000X

you see it in 10 seconds The new Polaroid 3000X Radiographic Packet is similar in speed and simplicity to the Polaroid Land films used for swift snapshots. It delivers a finished dry radiograph 10 seconds after exposure.

you take it with 80% less exposure The Polaroid 3000X Packet uses as little as 1/5 the exposure required for a conventional x-ray film: reduces radiation-dosage to patient by 80%.

plenty of latitude, too There's ample exposure leeway in the new Polaroid 3000X: no need for hair-line technique factors.

Polaroid packets, cassettes, and processors are now available for immediate delivery. Call any local Picker office or write Picker X-Ray Corporation, White Plains, N.Y.

Picker x-ray

MODERN SYSTEMS FOR X-RAY FILM PROCESSING

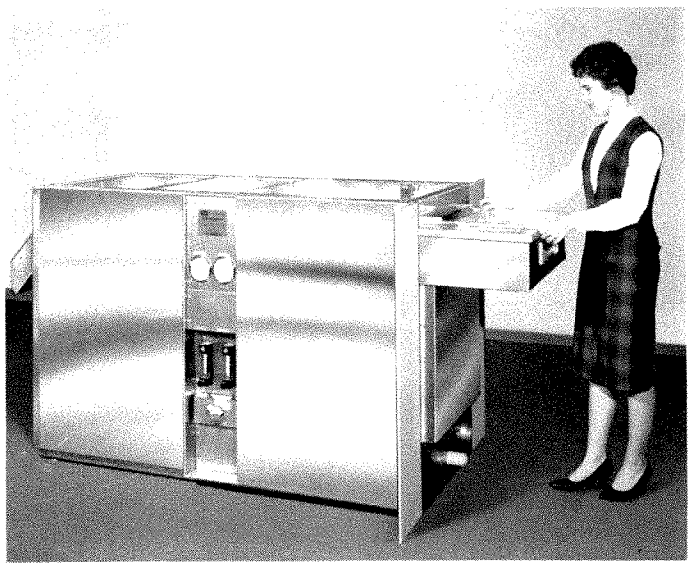
For AUTOMATIC Processing

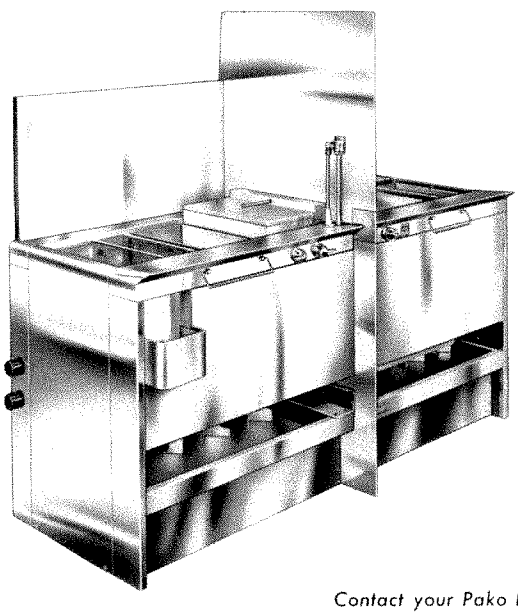
PAKO

Pakorol-X

Processor

The new Pakorol-X is a complete small, compact, low-cost system featuring fully automatic hangerless processing and drying. It's fast and quiet – 6½ minutes total processing and drying time for any brand of screen film. Satin finish stainless steel. AVAILABLE FOR RIGHT HAND OR LEFT HAND INSTALLATION.





For MANUAL Processing

PAKO

Model 39 X-Ray Filmtanx

The Model 39-X-Ray Filmtanx provides an efficient system for film processing. For cleanliness it's stainless steel throughout. Individual tanks with tempering cells and drain valves. Easy to get at control and plumbing connections. Sixty or more radiographs an hour. AVAILABLE FOR RIGHT HAND OR LEFT HAND THRU THE WALL INSTALLATION.

For consistent top quality control, Pako offers a complete line of associate products for chemical mixing and handling, circulation-filtration and automatic temperature control.

Contact your Pako Distributor, or write directly to Pako Corporation.

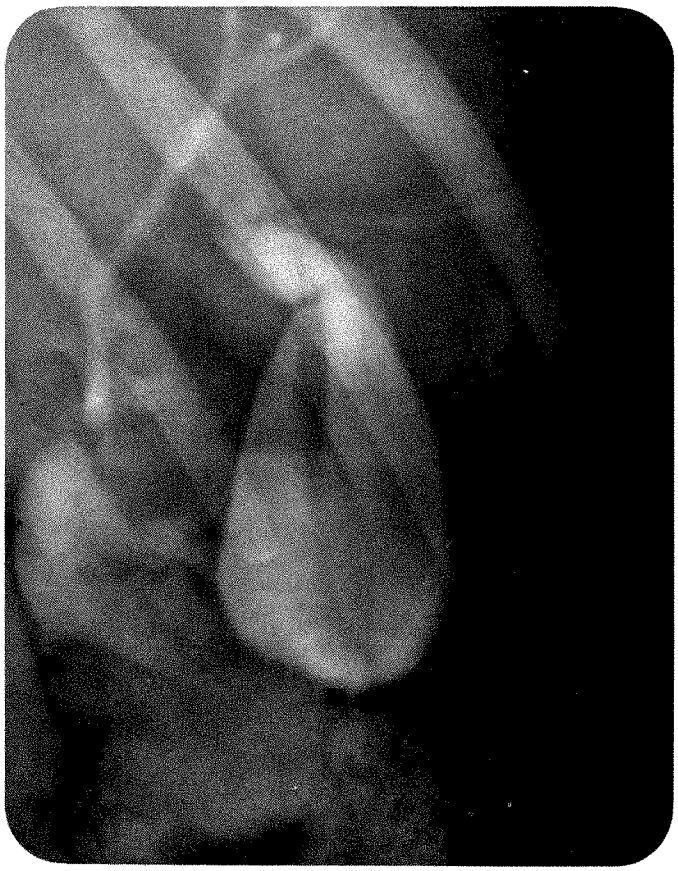
PAKO

PAKO CORPORATION

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PAKO FOR PROGRESS

"Outstanding Contribution"[1] in Cholangiography



Rapid Optimal Opacification: Within 15 minutes, Cholografin outlines hepatic and common ducts even after cholecystectomy, reveals biliary ducts in about 25 minutes, and completely opacifies the gallbladder within 2½ hours.[1-4]

High Diagnostic Accuracy: Accuracy in diagnosing bile duct disease in postcholecystectomized patients is reported to be 86%.[1] In another series,[5] Cholografin permitted diagnostic interpretation in almost 70% of patients with chronic cholecystitis.

"With Little Risk"[5] No hepatic or renal toxicity, no delayed reactions have been observed.[5] Cholografin "is the method of choice"[6] for gallbladder visualization in infants.

Cholografin
Sodium / Methylglucamine

Duografin Squibb Diatrizoate and Iodipamide Methylglucamines for rapid visualization of biliary *and* renal tracts in routine examinations or differential diagnosis

Supply: Cholografin Methylglucamine Squibb Iodipamide Methylglucamine Injection U.S.P. is supplied in 20 cc. sizes, with sufficient excess for sensitivity testing.

Cholografin Sodium Squibb Iodipamide Sodium Injection N.F. is supplied in cartons containing two 20 cc. ampuls with sufficient excess for sensitivity testing.

Duografin is supplied in bottles of 50 cc.

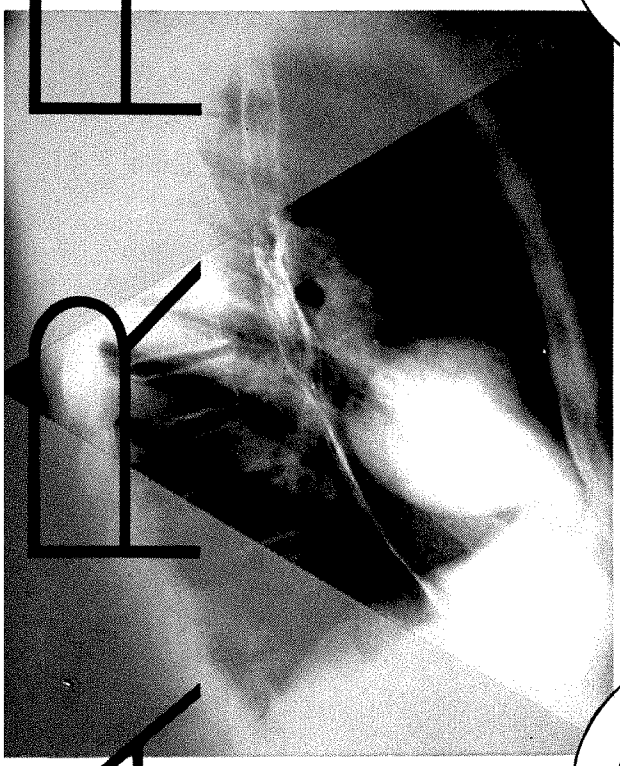
For full information, see your Squibb Product Reference or Product Brief.

References: (1) Cohn, E. M.: Am. J. Gastroenterol. *35*:115 (Feb.) 1961. (2) Jones, M. D.; Sakai, H.; and Rogerson, A. G.: J. Pediat. *53*:172 (Aug.) 1958. (3) Machella, T. E.: Gastroenterology *34*:1050 (June) 1958. (4) Orloff, T. L.: Am. J. Roentgenol. *80*:618 (Oct.) 1958. (5) Johnson, G., Jr.; Pearce, C.; and Glenn, F.: Ann. Surg. *152*:91 (July) 1960. (6) McClenahan, J. L.: Pennsylvania M. J. *62*:188 (Feb.) 1959.

'CHOLOGRAFIN'® AND 'DUOGRAFIN'® ARE SQUIBB TRADEMARKS.

SQUIBB
Squibb Quality — the Priceless Ingredient

SHARP







From large dense masses to confined areas difficult to reach, your GEVAERT medical x-ray film aids in producing an image of concise clarity and definition. And you achieve this maximum performance with a minimum of exposure.

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GEVAERT
X-RAY FILM

60-3M

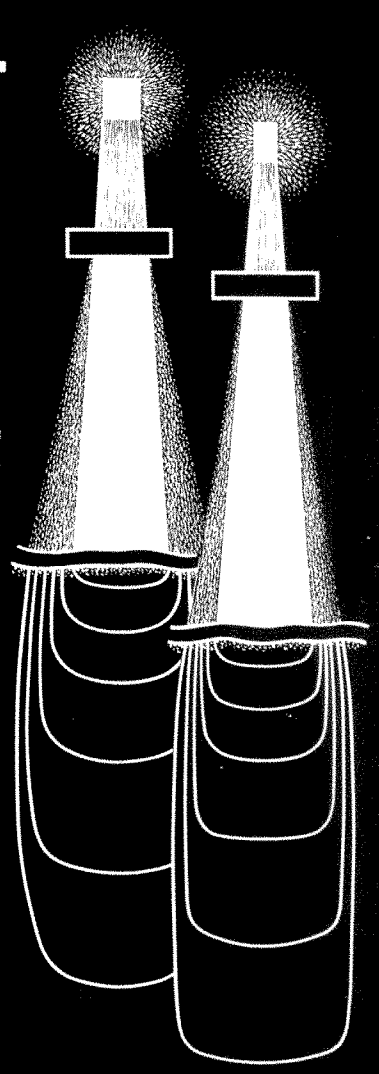
NOW – from A.E.C.L.
A NEW HIGH
IN COBALT 60 SPECIFIC ACTIVITIES

Here's what quantity production of Cobalt 60 of up to 150 curies per gram quality will mean in your COBALT CLINIC.

1 HIGHER OUTPUTS – Without penumbra problems – shorter treatment times even at increased treatment distances.

2 LESS PENUMBRA – With smaller source dimensions, sharper field definition, less integral dose, more beam control.

3 GREATER CHOICE OF SOURCE DIMENSIONS – Now clinically adequate outputs are available from smaller diameter sources than ever before. You can also have virtually "tailor made" sources in diameter increments of ¼ cm, between 0.5 and 3.0 cm for optimum size-output combinations.



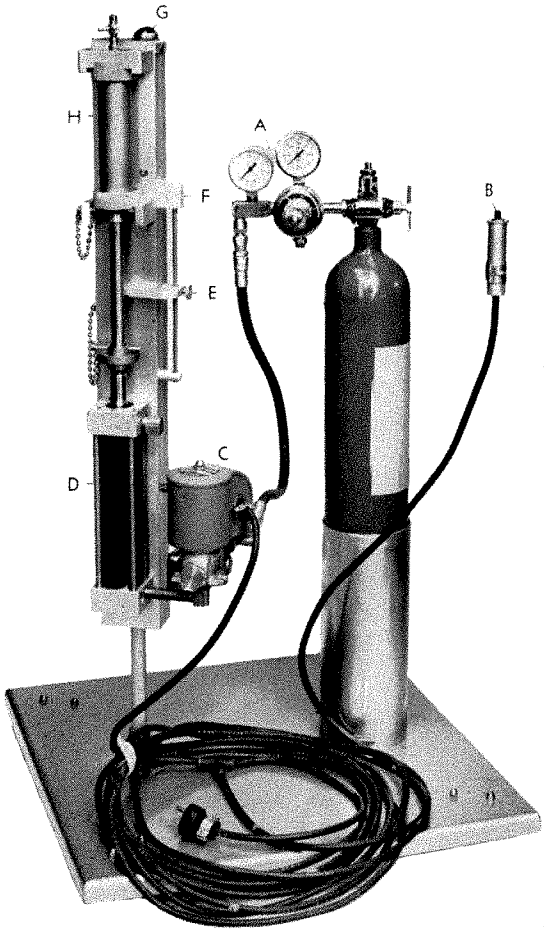
NOTE: **These new high output sources can be used in any AECL unit now that greatly increased capacities are available using AECL's new URANIUM shielding insert. (Uranium shielding inserts are available for all AECL Cobalt units, new or installed, providing maximum capacities of 3,000 curies to 8,000 curies plus!)**

Don't forget, your present source is always worth money under the **AECL SOURCE REPLACEMENT PLAN.**

For more information on high output sources telephone collect, or write and have our representative call.

ATOMIC ENERGY OF CANADA LIMITED
COMMERCIAL PRODUCTS DIVISION • P.O. BOX 93 • OTTAWA, CANADA

THE NEW PHELAN PHASIC DYE INJECTOR



is ready to help you by demonstrating its superior quality and simplicity of construction. Use this piece for thirty days and convince yourself that it is the best value on the market today in a phasic dye injector.

The component parts of the injector are:

- **A. Pressure regulator gauge.**
- **B. Momentary switch to energize solenoid valve.**
- **C. Solenoid switch operating on 115 V.**
- **D. Air cylinder—power source.**
- **E. Adjustment for graduating amount of media expressed.**
- **F. Micro switch to deenergize solonoid valve.**
- **G. Pilot light operating simultaneously with syringe.**
- **H. Syringe. 100 cc capacity furnished with unit. (50 cc capacity available at extra cost.)**

CO_2 gas recommended for use. *Never use oxygen.*

The complete unit is adjustable for height on its vertical standard. Mounted on steel base with baked enamel finish—supported by four conductive castors.

For further information write or phone collect

PHELAN MANUFACTURING CORP.
2523 Minnehaha Avenue Minneapolis 4, Minnesota

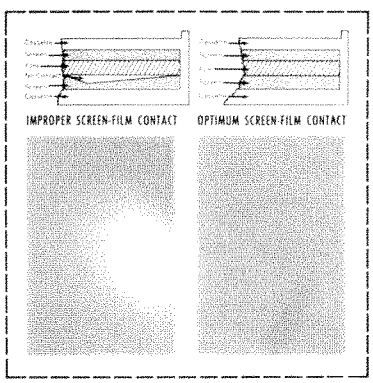
Now . . . A Major Advance in Modern Intensifying Screens



(Above) **Ansco Dura-Speed** extremely high-speed intensifying screens permit minimum patient exposure with maximum image quality with high KvP techniques. Also shorten exposure time and provide maximum detail with low capacity equipment.

(Above) **Ansco Dura-D** medium speed intensifying screens provide exceptionally fine detail at high or low KvP. Their fine grain structure is ideal for techniques requiring ultra-fine image detail. Well suited to general radiographic work.

Plus All These Other Advantages!



Better Screen-Film Contact (See illustration at left). The balanced density and thickness of Ansco's screen base material has *strength* to protect the delicate calcium tungstate layer from stress, yet is *soft* enough to allow the screen to conform to the cassette for optimum screen-film contact.

Easier Cleaning. No special cleaner needed. A damp cloth or mild soap and water solution easily removes most stains associated with screens.

Longer Life. The use of "chemical" pure materials in the manufacture of Ansco screens, plus their special non-pitting surface coating, assures long life with no recession in speed.

Consistent Quality. Ansco's exacting requirements for consistent grain size and layer thickness guarantee uniform speed ratings from pair to pair of screens.

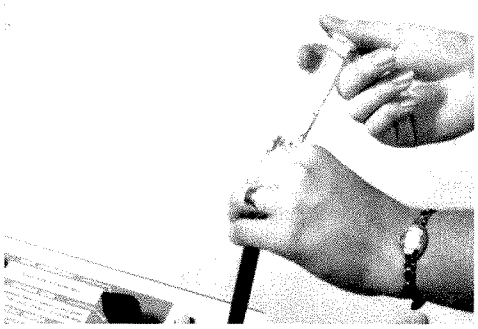
Static Resistant. All materials used in the manufacture of Ansco Intensifying screens are selected for their resistance to static electricity.

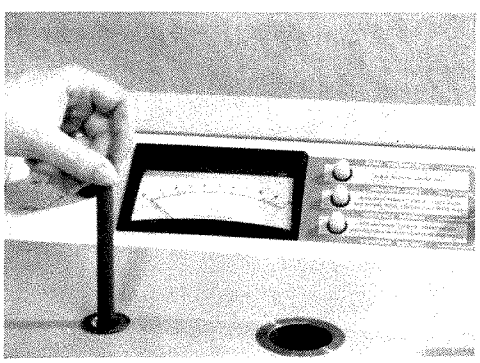
ANSCO, Binghamton, N. Y., A Division of General Aniline & Film Corporation

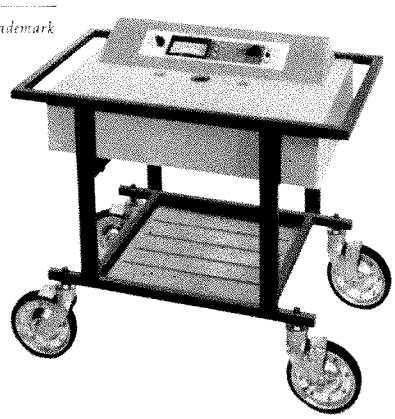
Ansco
X-ray



BLOOD VOLUME MEASUREMENT IS EASY!







With Atomium's new VOLĒMETRON,* you can quickly obtain highly accurate volume measurements of whole blood, plasma, or red cells. It's as easy as this:

1. Prepackaged syringe containing radioactive tracer material is placed in DOSE well of VOLĒMETRON. Value of dose is automatically stored in memory circuit.
2. Reference blood sample is drawn. The measured dose is administered and empty dose syringe returned to DOSE well. VOLĒMETRON automatically corrects its "memory," subtracting value of residue in the syringe and instrument background.
3. After sufficient mixing time, postmix blood sample is drawn. Both reference sample and postmix sample are placed in respective VOLĒMETRON wells.

Now, VOLĒMETRON automatically computes the patient's blood volume, indicating it directly in liters on an illuminated meter.

Total determination time: 10-15 minutes (including mixing of dose in patient's system). Accuracy for overall procedures: ±5% (even for consecutive determinations). *For full details, write Atomium Corporation, 940 Main Street, Dept. 40-B, Waltham 54, Massachusetts.*

European Office: 27 Alkmaarsestraat, Scheveningen, Holland

* *Trademark*

atomium

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Affiliate, The PERKIN-ELMER Corp.

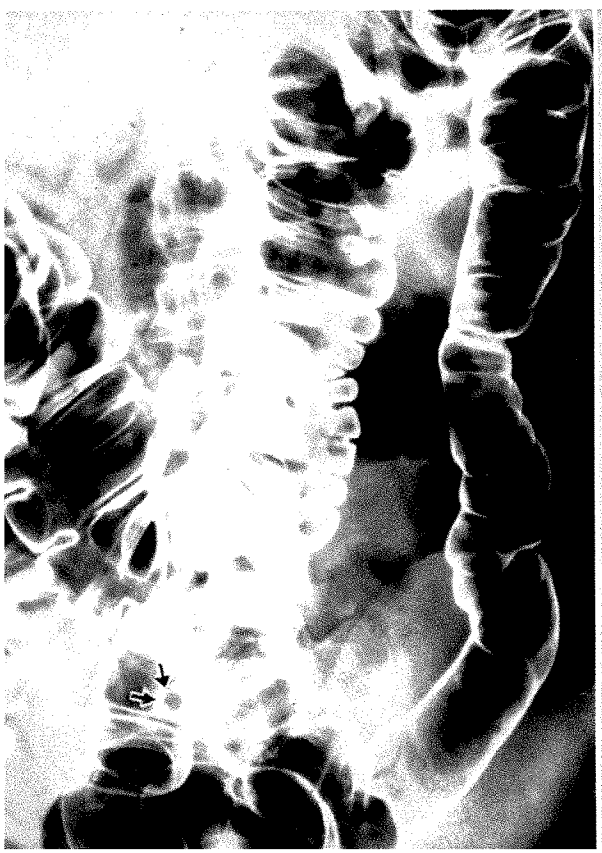
...radiological studies with Barotrast
are routinely exceptional...

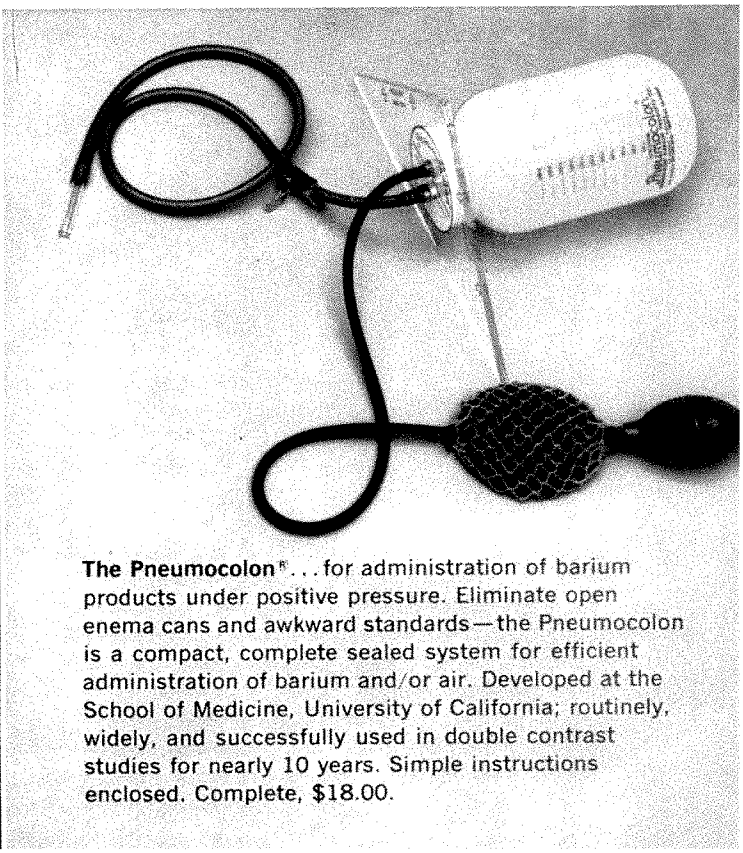
*BAROTRAST MAINTAINS UNIFORM SUSPENSION

Barotrast will not separate, crack, peel, or clump.
Its unique adherence to the intestinal mucosa gives a uniform, stable, and flexible coating. It possesses unparalleled properties of flow, coating, and water retention which are not affected by mucin content, enzymes, or changes in pH.
Recognizing these singular benefits of Barotrast, many radiologists who have already adopted its use for double contrast studies are now finding Barotrast to be exceptional in upper GI and small bowel studies.
Available in 25-lb. cartons through all radiological supply houses. Literature available from Barnes-Hind.

*A SPECIALLY PROCESSED MICROPARTICLE FORM OF BARIUM SULFATE U. S. P. . . . DEVELOPED AND MANUFACTURED BY

BARNES-HIND BARIUM PRODUCTS, INC., 895 KIFER ROAD, SUNNYVALE, CALIFORNIA.





The Pneumocolon®. . . for administration of barium products under positive pressure. Eliminate open enema cans and awkward standards—the Pneumocolon is a compact, complete sealed system for efficient administration of barium and/or air. Developed at the School of Medicine, University of California; routinely, widely, and successfully used in double contrast studies for nearly 10 years. Simple instructions enclosed. Complete, $18.00.

The contrast medium that is ideally opaque

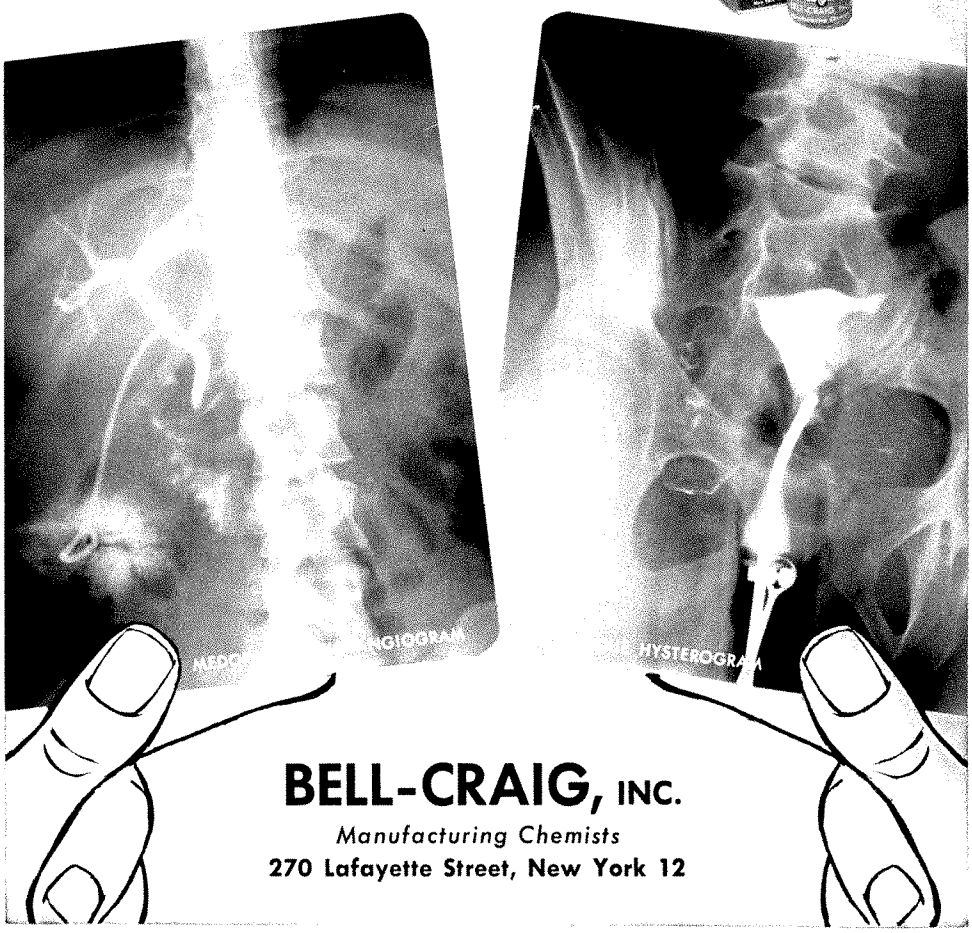
for operative cholangiography and hysterosalpingography

Not too dense as to mask pathologies. Well tolerated, completely absorbable, suitably viscous, aqueous and miscible, more and more Medopaque is becoming the contrast medium of choice.

MEDOPAQUE is an aqueous, sterile, viscous contrast solution containing 35% Sodium Ortho-iodohippurate, 10% Sodium Iodide and a blend of polyethylene glycols. Net iodine content 20%.

MEDOPAQUE





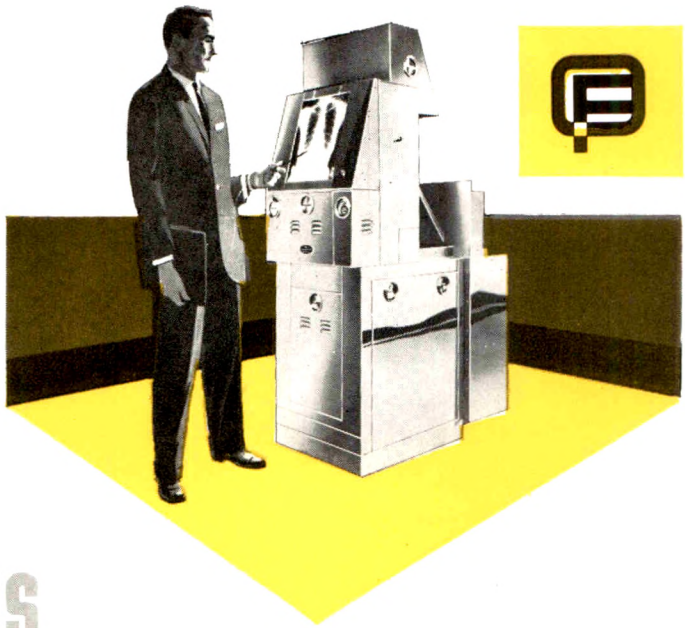
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requires only 8½ sq. feet of floor space



PROCESSES SCREEN AND NONSCREEN FILM IN MIXED SIZES, AT 150 PER HOUR

. . . . but Fax-Ray Junior's capabilities do not end here. This versatile little unit will also process industrial films and X-ray papers with equal fidelity and trouble free operation.

In the FAX-RAY JUNIOR, continuous control over processing factors is maintained. Processing speeds are adjustable to variable constants, temperatures of chemical solutions and wash-waters are automatically controlled to within 1/4°F and films are transported automatically through a processing system of high impingement agitation of continuously filtered and recirculated solutions for maximum resolution and grain refinement.

There is no film loss or damage. Finished films are delivered dry and retain original exposure characteristics to the highest degree of fidelity and clarity.

A few of the many desirable features to be found in the Fax-Ray Junior are:
Convenient, self contained viewer for "on the spot" emergency readings — constructed of type 316L Stainless Steel — required maintenance time cut to minutes per day — processes any manufacturer's film in mixed sizes up to 14" X 17" — uses standard solutions — installs through the wall or within the darkroom.

Fax-Ray Junior sells COMPLETE for $7500. * Write for complete information.

* MANUFACTURER'S SUGGESTED SELLING PRICE. SUBJECT TO CHANGE WITHOUT NOTICE.

FAX-RAY JUNIOR ®

OSCAR FISHER COMPANY, INC. ◆ *Newburgh, N. Y.,* U.S.A.



mfgrs. of:

anhydrator ®

DRIES FILM WITHOUT HEAT IN LESS THAN TEN MINUTES!!!

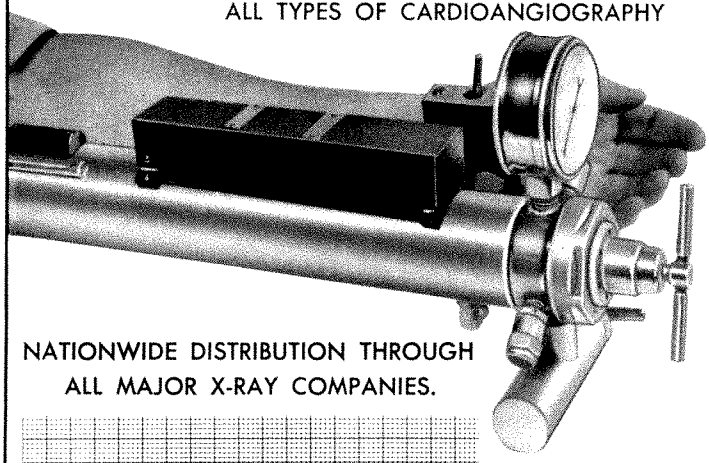


SPRAY **processall** ®

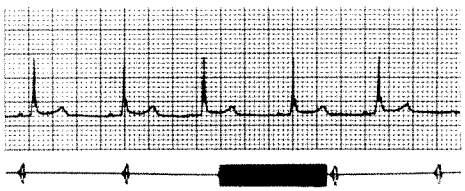
HIGH GAMMA, AUTOMATIC ROLL FILM PROCESSOR

AMPLATZ INJECTOR

A COMPACT PRESSURE DEVICE FOR
ALL TYPES OF CARDIOANGIOGRAPHY



NATIONWIDE DISTRIBUTION THROUGH
ALL MAJOR X-RAY COMPANIES.



- ✓ 36 cc of 75% Hypaque thru No. 7 catheter in 1 second
- ✓ hand operated or remote control
- ✓ portable and compact.
- ✓ only 10 pounds in weight.
- ✓ safe and reliable, extensively tested.
- ✓ simple in operation.
- ✓ unique low friction stainless steel syringe.
- ✓ effective pressure control to 750 or 1200 psi.
- ✓ automatic exposure release.
- ✓ thermostatically controlled heating jacket.
- ✓ injection indicator for EKG.
- ✓ powered by CO_2 cartridge.

NEDMAC, INC.
SCIENCE INDUSTRY CENTER
MINNEAPOLIS 27, MINNESOTA

A HISTORY OF THORACIC SURGERY

RICHARD H. MEADE, B.S., M.D.
Formerly on the Faculty of
University of Virginia Medical School
University of Pennsylvania Medical School
Northwestern Medical School

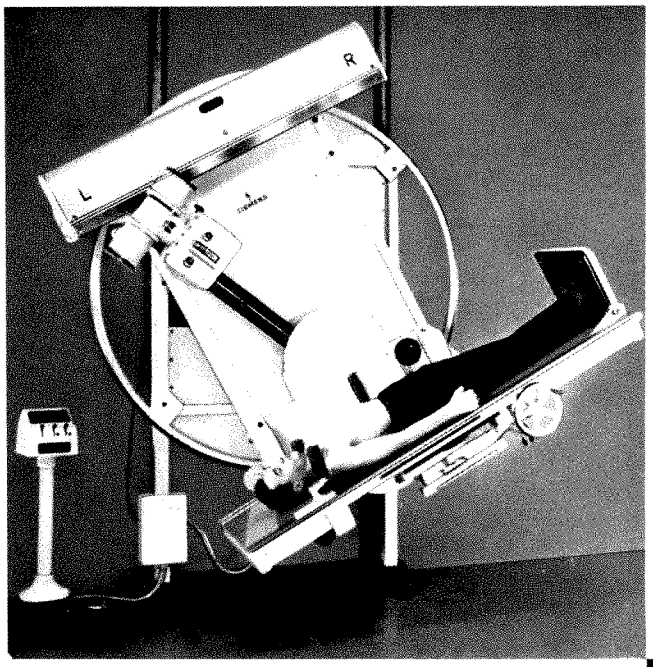
At last the literature includes a fascinating and completely comprehensive history of thoracic surgery—*covering the subject from the time of the Smith Papyrus to the present.* Although the period before 1900 was important in the evolution of thoracic surgery, the really dramatic advances have come since that date. Dr. Meade has known the men who made history in this field.

So interesting are the events recorded here that the book should appeal to every doctor regardless of his special interests. It will be of tremendous value to the man interested in problems of the chest. *In some respects it is less a history and more a textbook giving the development of surgical procedures, embellished by instructive details including results.* It could profitably be consulted in the care of surgical disorders of the chest.

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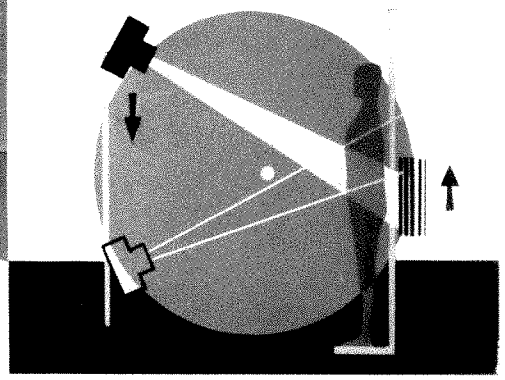
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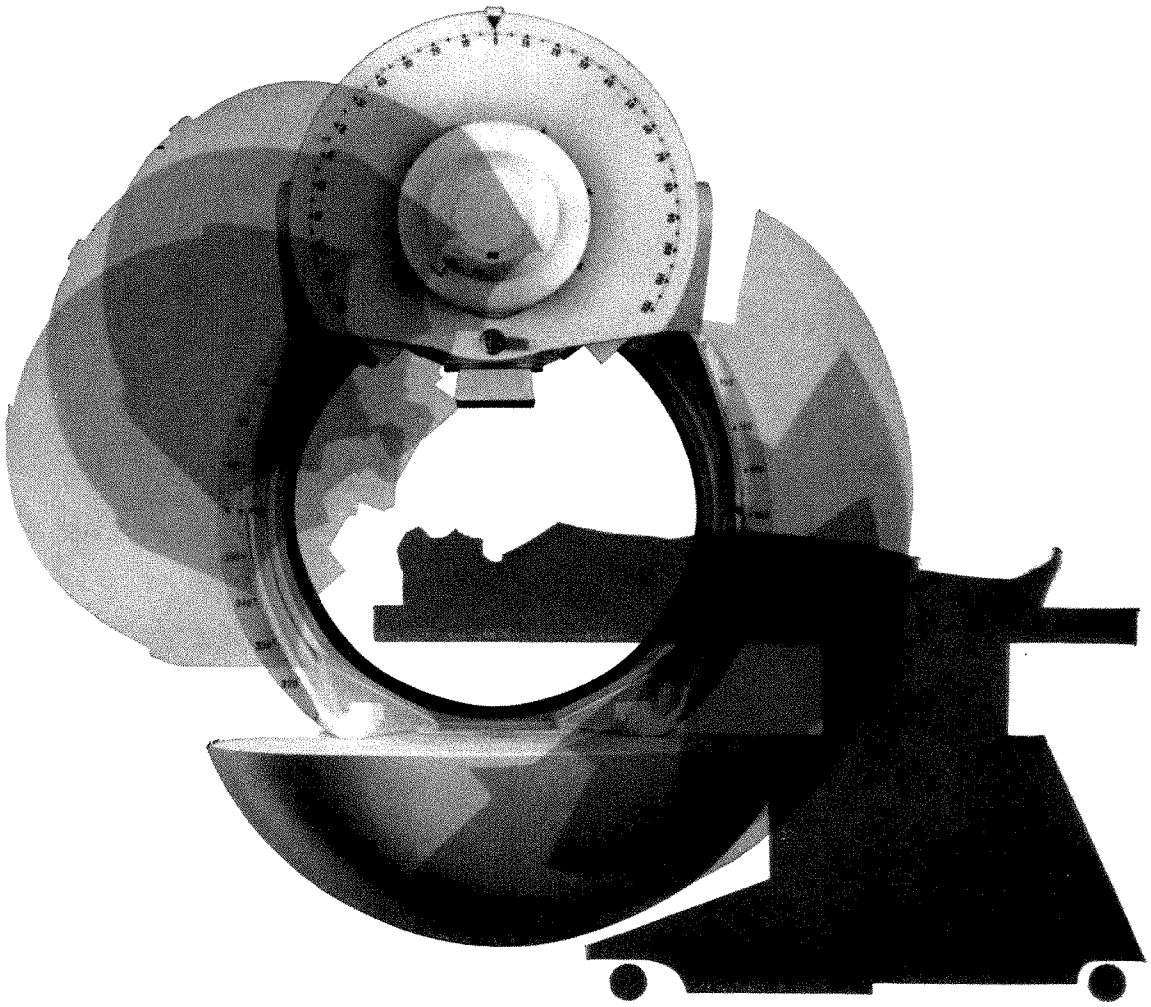
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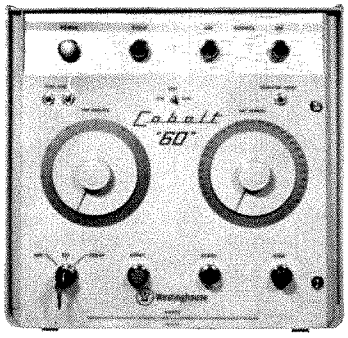
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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 86 AUGUST, 1961 NO. 2

THE OCCIPITO-ATLANTO-AXIAL JOINTS IN RHEUMATOID ARTHRITIS AND ANKYLOSING SPONDYLITIS*

By WILLIAM MARTEL, M.D.
ANN ARBOR, MICHIGAN

THE occipito-atlanto-axial region has special significance in "rheumatoid disease"* because it contains synovial tissue and important ligaments closely arranged within a small, critical space. Nontraumatic, atlanto-axial dislocation has been reported in ankylosing spondylitis and also in rheumatoid arthritis,[3,6,14,17,18,21,22,32] but the prevalence and significance of anterior displacement of the atlas in these conditions have not been generally appreciated. At times, anterior dislocation of the atlas was the initial manifestation of rheumatoid disease[31,44] and occasionally its relationship to coexisting chronic arthritis was apparently not recognized.[33,38] Furthermore, only a few reports have emphasized that the cervical spine may be affected not only in children but also in adults with classic rheumatoid arthritis.[5,10,22,37]

Neck pain and stiffness are not uncommon in rheumatoid disease. In view of the frequency of peripheral joint subluxations in such patients, it was of interest to obtain flexion views of the cervical spine to learn whether these joints were similarly affected. Subluxations, especially atlanto-axial and vertebral erosions, particularly of the odontoid process, were common. A vertical displacement of the cervical spine with respect to the foramen magnum was a frequent finding.

ANATOMY

The odontoid process forms median synovial joints anteriorly with the atlas and posteriorly with the transverse ligament (Fig. 1). This ligament is responsible for the stability of the atlanto-odontoid joint and prevents the atlas from slipping forward when the head is flexed (Fig. 2). It may contain a cartilaginous body against which the dens articulates, and a synovial bursa may be found behind it. The atlanto-axial joints function chiefly in rotation but

* The term "rheumatoid disease" is used here in a general sense to include rheumatoid arthritis, ankylosing spondylitis and the so-called rheumatoid variants, such as juvenile rheumatoid arthritis and arthritis associated with psoriasis or with chronic idiopathic ulcerative colitis.

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.
From the Department of Radiology, The University of Michigan, Ann Arbor, Michigan.

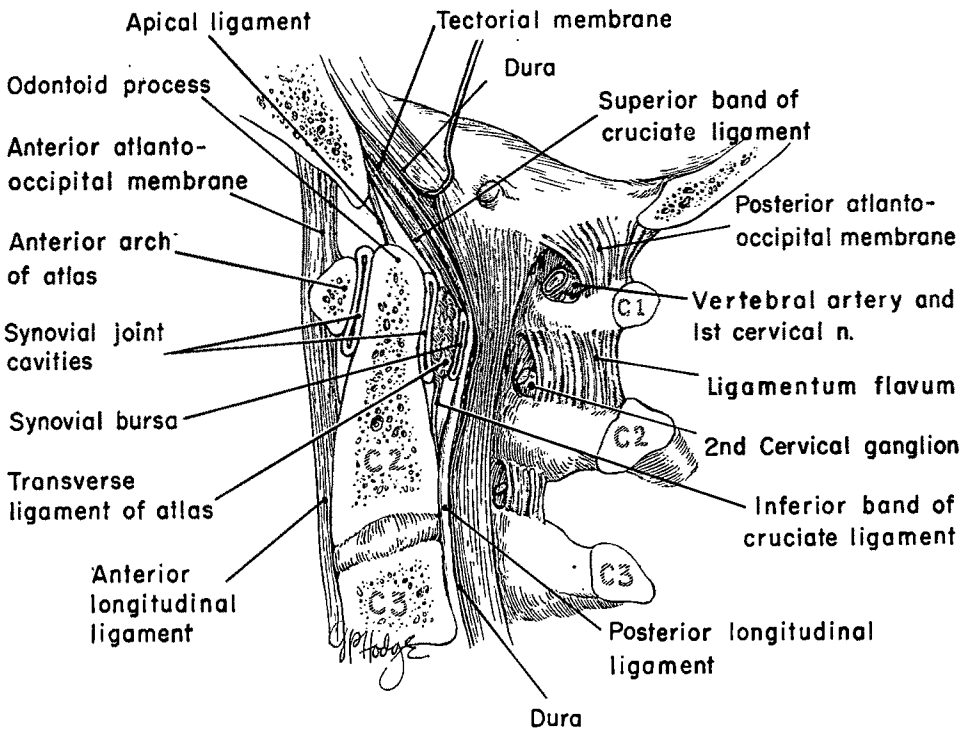


FIG. 1. Mid-sagittal section. Relationship of synovial tissue and ligaments to the atlas, odontoid and foramen magnum. An inconstant synovial bursa may also be found beneath the anterior margin of the foramen magnum.

normally also provide a minimal degree of flexion-extension motion. Thus, a cranial shift of the anterior atlas relative to the dens can be noted in extension and a slight gaping of the superior aspect of the atlanto-odontoid space may be seen in flexion.[44] For practical purposes, this space is constant in adults in flexion and extension, especially inferiorly, and is said not to exceed 2.5 mm.[16] It is apparently impossible to separate the odontoid from the atlas in cadavers unless the transverse ligament is completely cut.[44] In such instances the alar ligaments[44] and possibly also the external occipito-atlanto-axial ligaments[27] limit the degree of forward displacement of the atlas and skull.

The tectorial membrane and the external cranio-vertebral ligaments have been regarded as limiting the range of flexion-extension of the head, a motion which occurs chiefly in the atlanto-occipital joints. Werne[44] believes that flexion in these joints may normally be checked by skeletal contact between the anterior margin of the foramen magnum and the apex of the dens via an interposed synovial bursa. Trolard[42] also noted an inconstant supra-odontoid bursa beneath the anterior margin of the foramen magnum. An anomalous median "third condyle" on the inferior aspect of the basilar process, sometimes bearing an articular facet, has been described.[30] This may represent a "manifestation of an occipital vertebra."[4]

Basilar invagination may be congenital or acquired and implies an upturning of the foramen magnum margins associated with a vertical shift of the spine with respect to the skull. This condition is suggested when the odontoid tip lies more than 4.5 mm. above McGregor's base line, from the hard palate to the inferior margin of the occipital curve.[26] Another, more direct index of basilar invagination is the angle subtended by the clivus and plane of the foramen

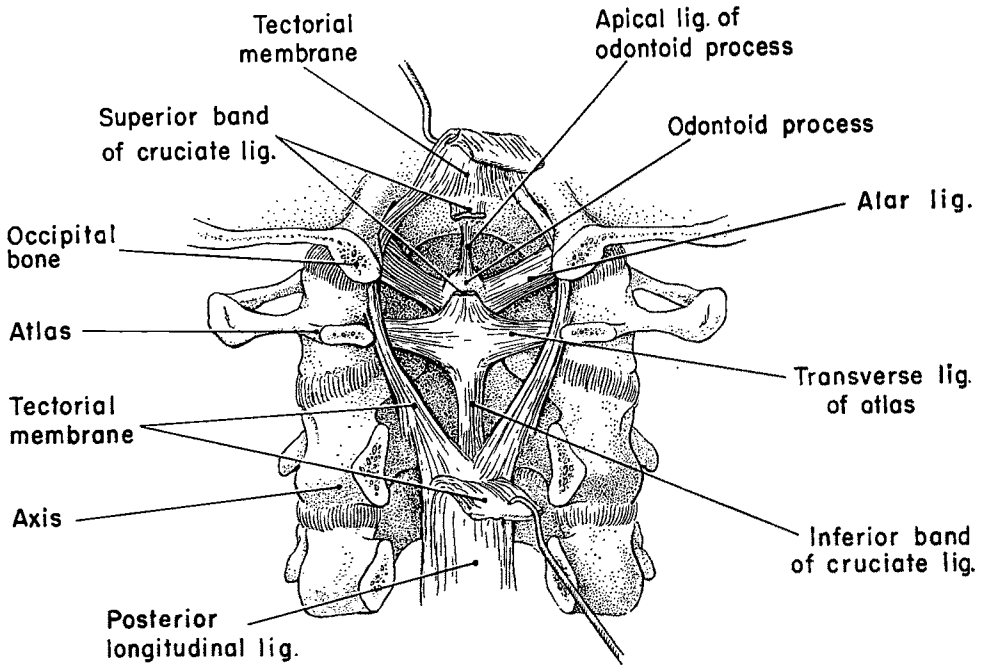


FIG. 2. Coronal section. Posterior view. The transverse ligament keeps the odontoid closely applied to the anterior atlas. (Synovial tissue and vertebral arteries are not shown.)

magnum which normally is said not to exceed 136°.[2,28]

METHODS AND MATERIAL

Lateral roentgenograms of the cervical spine were obtained in flexion and extension in 34 patients with rheumatoid arthritis and 32 with ankylosing spondylitis who were selected because of persistent neck discomfort or severe, progressive disease. Lateral midline laminagrams of the cervical spine were made in 60 of these patients and in 20 cases either open-mouth views of the odontoid process or frontal laminagrams were also obtained. The lumbosacral spine was examined in all but one patient with rheumatoid arthritis. Almost all with rheumatoid arthritis and approximately one-third of those with ankylosing spondylitis were current patients at the University Hospital. The others had been examined during the last four years and were recalled specifically for this study because neck discomfort had been a significant symptom. Three patients with rheumatoid arthritis and one with ankylosing spondylitis were included because a cervical subluxation was clinically suspected or known to have been present. Ankylosing spondylitis was differentiated from rheumatoid arthritis on clinical grounds[8,12] and on the basis of characteristic roentgen findings in the lower spine and pelvis in this disease.[34]

RESULTS*

The clinical data are summarized in Table 1. None of these patients were less than nineteen years of age, although 2 had the onset of rheumatoid arthritis at ages two and fourteen, and one had ankylosing spondylitis at the age of fifteen.

In these patients with rheumatoid arthritis, the disease was of long duration with persistently active synovitis and progressive joint deformities unappreciably altered by any therapeutic measures. Many had excessive and sometimes spectacular periarticular bone resorption, widespread rheumatoid nodule formation and strongly positive tests for the rheumatoid factor.

* Some of these cases have been previously reported.[22]

TABLE I

CLINICAL DATA

	Rheumatoid Arthritis	Ankylosing Spondylitis
Males	8	25
Females	26	7
Totals	34	32
Age (yr.)		
<30	3	3
30–39	3	3
40–49	9	16
50–59	13	6
>59	6	4
Duration (yr.)		
<10	10	3
10–20	14	14
>20	10	15
Rheumatoid Nodules	17	0
Rheumatoid Factor		
No. positive	21	4
No. tested	30	13
Steroid Therapy		
3 mo.–1 yr.	8	2
>1 yr.	23	3
Peripheral Joint Classification*		
Stage I	0	0
Stage II	2	0
Stage III	23	2
Stage IV	9	4
	34	6

* American Rheumatism Association criteria.[40]

Neck discomfort was or had been present in most cases, but in some instances of severe cervical instability this was not a prominent symptom. Subluxations of the peripheral joints were common and at least 7 patients had spontaneous tendon ruptures. The disease was also severe in the majority of patients with ankylosing spondylitis and many had virtually complete spinal rigidity. Almost all had neck discomfort and 14 patients received roentgen therapy to the cervical spine. Eleven of the 32 patients had hip or shoulder involvement and in 6 others the peripheral joints were affected as well.

The cervical spine roentgen findings are summarized in Table II.* Erosions of the odontoid cortex were present in approximately one-third of the patients with rheumatoid arthritis, but were less frequently demonstrated in ankylosing spondylitis. Bony ankylosis of the odontoid to the anterior atlas or basilar process, present in at least 7 patients, may have precluded the demonstration of such erosions in the latter group. There was an associated reactive sclerosis of the odontoid ("shining odontoid") in 3 of the 4 patients with ankylosing spondylitis who exhibited such surface erosions. Reactive sclerosis of the odontoid was apparent in one other patient with spondylitis and in one case of rheumatoid arthritis; in the latter the body of the axis was also sclerotic and eroded. There were cervical vertebral erosions other than in the odontoid in 9 patients with classic

TABLE II

CERVICAL SPINE ROENTGENOGRAPHIC FINDINGS IN 66 ADULTS

	Rheumatoid Arthritis	Ankylosing Spondylitis
Patients	34	32
Odontoid Erosions	12	4
"Shining Odontoid"	1(?)	4
Atlanto-Odontoid Distance (normal limit, 2.5 mm.)		
3–5 mm.	13	5
>5 mm.	11	0
Other Cervical Subluxations	10	4
Baseline-Odontoid Distance (normal limit, 4.5 mm.)		
5–8 mm.	5	7
>8 mm.	5	2
Foramen Magnum-Clivus Angle 136° or Greater	4	1

* Measurements given are uncorrected for roentgenographic enlargement. The correction factor varied (83–87 per cent), depending on the spine-film distance. The target-film distance was 60 inches for conventional roentgenograms and 58 inches for laminagrams.

rheumatoid arthritis. These were located in the vertebral end plates in 4 instances and at the tips of the spinous processes, usually C6, C7 or T1, in 8. Of these 9 patients, 4 had a normal odontoid process.

Abnormal atlanto-odontoid separation was present in 29 patients and, of these, flexion views were required for the diagnosis in 23. Of the 11 patients in which this was severe (greater than 5 mm.), 8 had no other cervical subluxations. Cervical subluxations other than atlanto-axial were frequent and were multiple (exclusive of C1–C2) in 5 patients with rheumatoid arthritis. These figures do not include instances of minimal malalignment. There were no cases of ankylosing spondylitis with multiple cervical subluxations.

Two patients with rheumatoid arthritis exhibited ankylosis of the posterior vertebral joints but neither had paraspinal ossifications and the disease began in childhood in both; one of these had bony ankylosis of the odontoid to the anterior atlas and basilar process. In another patient with rheumatoid arthritis who had severe, immobile anterior subluxation of the atlas of two years' duration, there was a suggestion of calcium deposits in the soft tissues of the atlanto-odontoid space and minimal periosteal new bone formation of the odontoid tip, but no bony ankylosis.

Upward displacement of the spine was common in both conditions. At times this was severe, especially in rheumatoid arthritis, and was associated with a wide foramen magnum-clivus angle with significant frequency.

REPORT OF CASES

RHEUMATOID ARTHRITIS

Case I (R. K. No. A2935).† A sixty year old male, who had moderately severe rheumatoid arthritis for twenty-two years, complained of recent neck pain and weakness. He received steroid therapy during the preceding months because of an exacerbation of his peripheral joint manifestations. Generalized lymphadenopathy and severe limitation of neck motion were present. There was a tender, subcutaneous nodule in the occiput. Multiple peripheral joints showed chronic, active synovitis. Neurologic examination was negative except for muscular weakness. The latex fixation test for the rheumatoid factor was strongly positive (1:5120). A roentgenogram showed an anterior subluxation of the atlas in flexion (Fig. 3). The sacroiliac joints were normal. He developed a left hemiplegia and expired. Cerebral arteriography had revealed occlusion of the right internal carotid artery. At necropsy the atlanto-axial subluxation was confirmed and bilateral cerebral infarcts were demonstrated. Microscopic examination of the odontoid process showed erosions of its cartilage and cortex, associated with ingrowth of proliferating pannus (Fig. 4*A*) and an adjacent, chronic, nonspecific synovitis. Ligaments adjacent to the odontoid showed areas of fibrinoid necrosis with chronic inflammation (Fig. 4*B*).

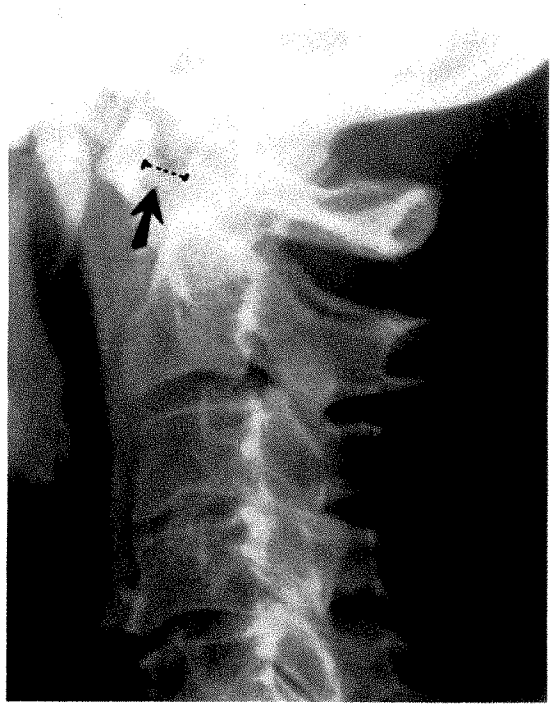


Fig. 3. Case I. Rheumatoid arthritis. Lateral view in flexion. The atlanto-odontoid distance (arrow) measured 7 mm. (Courtesy of *Arthritis and Rheumatism.*)

Case II (D. S. No. 756575). A fifty-four year old male had severe rheumatoid arthritis of multiple peripheral joints since 1948 for which

† From the Veterans Administration Hospital, Ann Arbor, Michigan.

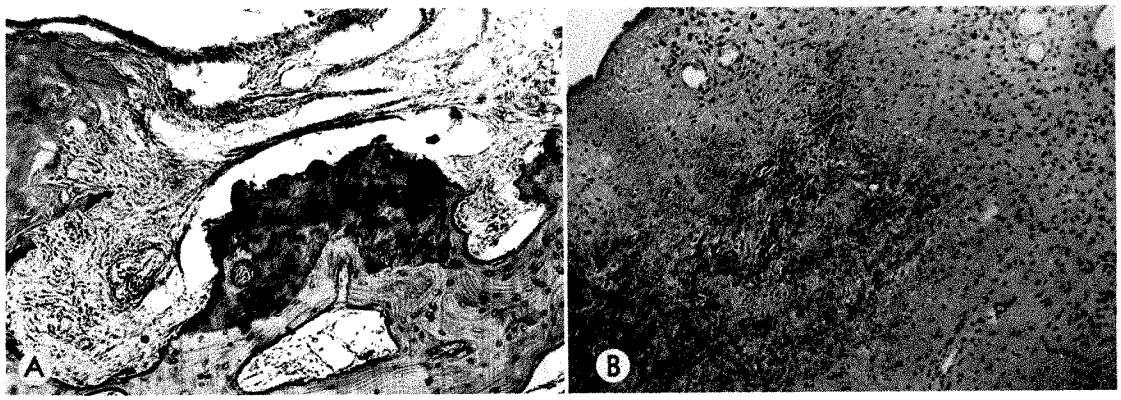


Fig. 4. Case I. Rheumatoid arthritis. (*A*) Odontoid process, ×90. Erosion of cartilage (upper left) and bone, with adjacent pannus. (*B*) Ligament which was adjacent to the odontoid, ×140. Fibrinoid necrosis and chronic inflammation.

he received steroid therapy for a period of eight years. In 1959 he developed neck pain and occipital headaches which were aggravated by head flexion. Neck motion was limited and there was tenderness over the upper cervical spine. He had numerous subcutaneous rheumatoid nodules and multiple extensor digitorum communis tendon ruptures. Neurologic examination was negative. The latex fixation test was positive (1:5120). Cervical roentgenograms and a midline laminagram showed normal vertebral alignment in a neutral position but anterior dislocation of the atlas in flexion; the atlanto-odontoid distance measured 11 mm. The odontoid process was severely eroded and erosions were also present in the vertebral end plates of C6 (Fig. 5, *A*, *B* and *C*).

Case III (J. P. No. 433316). Rheumatoid arthritis began at the age of twenty-one in this

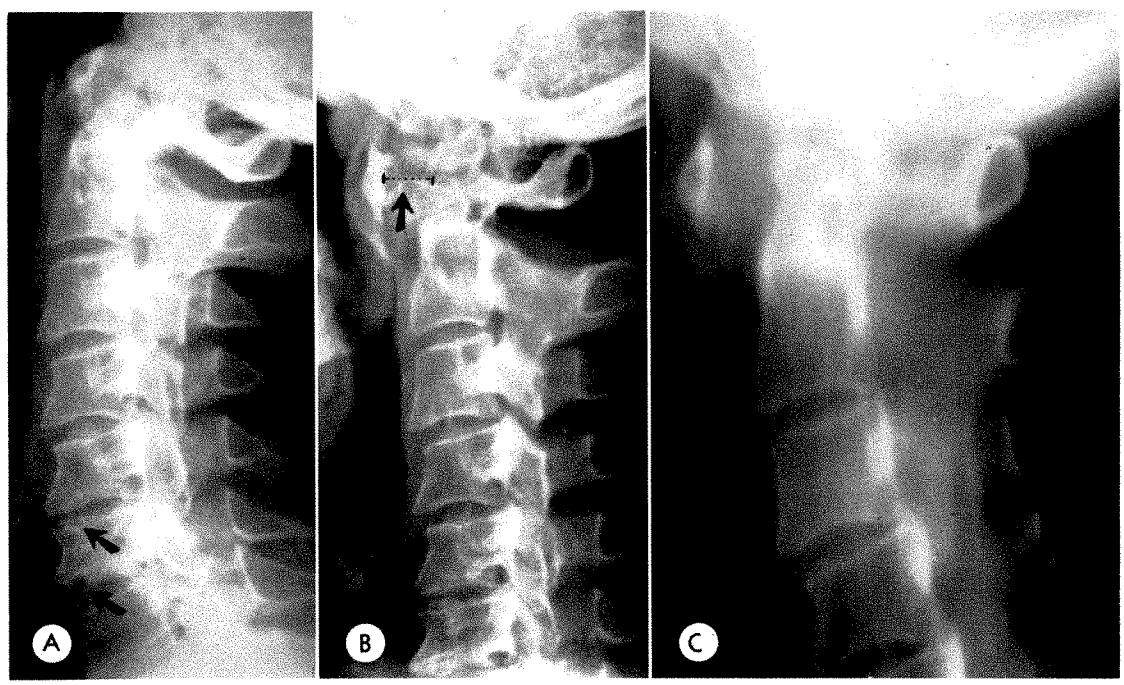


Fig. 5. Case II. Rheumatoid arthritis. (*A*) Extension. Normal atlas alignment. Erosions of C6 (arrows). (*B*) Flexion. Atlanto-odontoid separation of 11 mm. (arrow). (*C*) Midline laminagram. Erosion of the odontoid process. (Courtesy of *Arthritis and Rheumatism*.)

forty-three year old male, but was relatively quiescent until 1958. At that time, after an episode of pneumonia, he developed severe, persistent synovitis of virtually all peripheral joints and was treated with steroids for several weeks. Physical examination in 1960 showed advanced rheumatoid arthritis of many joints and multiple rheumatoid nodules. He had a nasopharyngeal soft tissue mass which proved to be lymphoid hyperplasia. Neurologic findings were negative except for weakness and a gross action tremor which he said began in 1958. The latex fixation test was positive (1:5120). A roentgenogram showed an increase in the atlanto-odontoid distance from 1 to 3 mm. with flexion and an irregular odontoid erosion at the point of articulation with the transverse ligament (Fig. 6).

Case IV (L. B. No. 934431). A nineteen year old male had episodes of migratory polyarthritis at ages fourteen and seventeen. He developed fever, persistent arthritis of several peripheral joints, and neck stiffness two years previously for which he received steroid therapy. Physical examination showed neck motion to be limited. The heart was normal. There was active synovitis in multiple joints. Serologic tests for the rheumatoid factor were negative. Roentgenograms showed bony ankylosis of several cervical apophyseal joints. In flexion the atlanto-odontoid distance increased to 4 mm. He had typical basilar invagination (Fig. 8, *A* and *B*). The odontoid-base line distance was 15 mm. and the foramen magnum-clivus angle was 136°. The sacroiliac joints were normal and there were no paraspinal ossifications.



Fig. 6. Case III. Rheumatoid arthritis. Midline laminagram. Irregular erosion of the posterior surface of the odontoid at the articulation with the transverse ligament. Note nasopharyngeal lymphoid hyperplasia and prominence of retro-odontoid soft tissue.



Fig. 7. Rheumatoid arthritis. Midline laminagram showing an erosion of the posterior aspect of the odontoid with an adjacent bone fragment resembling osteochondritis dissecans. There are multiple cervical subluxations.

Case V (C. S. No. 731351). A forty-two year old female physician had severe, progressive rheumatoid arthritis of virtually all peripheral joints since the age of twenty-four. There was widespread destruction of articular cartilage and bone, especially in the hands and wrists.

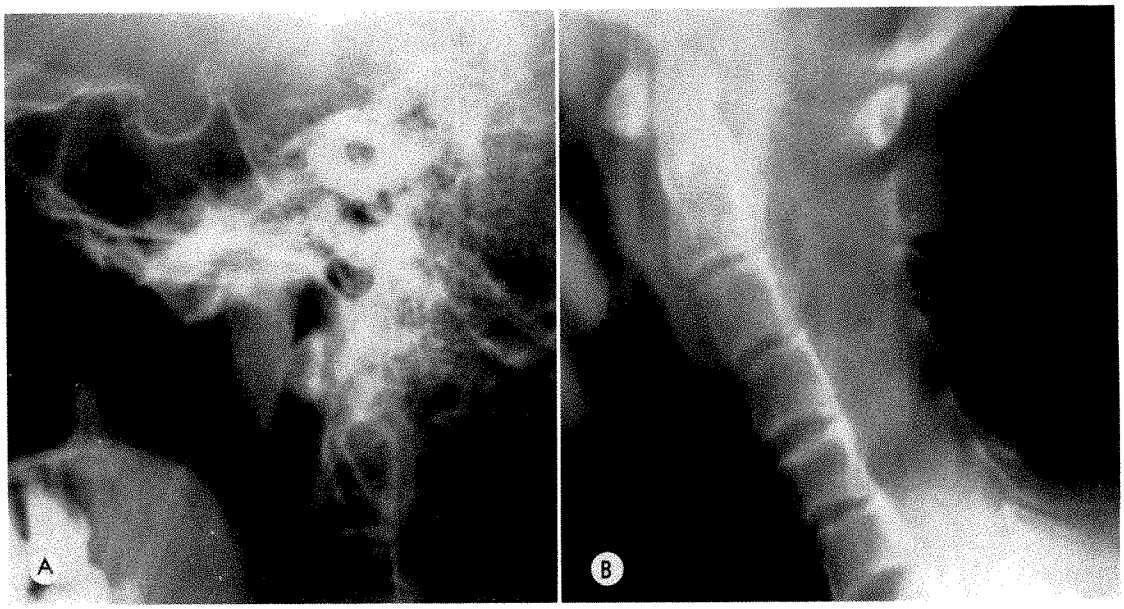


Fig. 8. Case iv. Rheumatoid arthritis. (*A*) Lateral view. (*B*) Midline laminagram. Typical basilar invagination and deformity of the vertebral bodies which was associated with ankylosis of the posterior vertebral joints.

She had multiple rheumatoid nodules, lymphadenopathy and splenomegaly. There was no significant limitation of spinal motion. Neurologic findings suggested peripheral neuritis. Serologic tests for the rheumatoid factor were repeatedly positive. A roentgenogram showed multiple cervical subluxations with decrease in the height of the intervertebral disks and erosions of the vertebral end plates, posterior arches, articular processes and odontoid. The latter extended 12 mm. above McGregor's base line and the foramen magnum-clivus angle measured 142°. There was a soft tissue density behind the odontoid which was felt to represent inflammation or brain tissue extending into the spinal canal (Fig. 9).

Case vi (C. T. No. 256613). A fifty-six year old male had severe, progressive rheumatoid arthritis since 1948 with involvement of virtually all peripheral joints. He had marked periarticular bone resorption in the hands and wrists. There were no significant symptoms referable to the spine and no manifestation of spinal cord compression. Serologic tests for the rheumatoid factor were persistently positive. Roentgenograms showed multiple cervical subluxations with erosions of the posterior arches and articular processes. The axis was also eroded and sclerotic and the atlas had descended to its inferior margin; the odontoid process was sclerotic and extended into the posterior fossa. The foramen magnum-clivus angle measured 136° but the upturning of the foramen magnum margins, typical of severe basilar invagination, was absent (Fig. 10, *A* and *B*).

ANKYLOSING SPONDYLITIS

Case vii (J. G. No. 528883). A thirty-one year old male had the onset of arthritis of the sacroiliac joints, hips and knees at the age of fifteen. Spinal and costovertebral joint involvement was progressive with severe limitation of motion and reduction in chest expansion. Pain due to cervical spondylitis (Fig. 11*A*) necessitated roentgen therapy in 1954 with some relief. He was unable to rotate his head on physical examination in 1960. Roentgenograms showed that the atlas had become dislocated anteriorly 5 mm. A laminagram demonstrated bony proliferation of the odontoid with fusion to the atlas in the dislocated position (Fig. 11*B*). He also developed an erosion of the spine of C7 during this six year interval. There was complete bony ankylosis of the sacroiliac and posterior vertebral joints with paraspinal ossifications, characteristic of ankylosing spondylitis.

Case viii (N. R. No. 327511). A sixty-one

year old female developed ankylosing spondylitis in 1943. Her spine became progressively rigid and chest expansion was reduced. The peripheral joints were normal. She complained of neck pain (Fig. 12*A*) and stiffness for several years for which she received roentgen therapy (250 r in 1945) and she also noted a cracking sensation on head motion. Roentgenograms and a midline laminagram (Fig. 12*B*) in 1960 showed superficial erosions of the odontoid process, associated with a reactive sclerosis and mottling of the trabecular pattern. The sacroiliac joints and lumbar spine showed characteristic changes of ankylosing spondylitis.

Case ix (E. S. No. 713526). Ankylosing spondylitis and psoriasis developed at the age of thirty-four in this forty-six year old male. His spine became almost totally rigid and chest expansion was severely limited. He had arthritis of almost all peripheral joints. There were no subcutaneous nodules and the serologic tests for the rheumatoid factor were repeatedly negative. Neurologic examination was difficult because of extensive psoriasis and widespread ankylosis of the peripheral joints. Comparative roentgenograms made in 1952 and 1959 showed that the anterior arch of the atlas had become reduced in thickness and that the odontoid had also become small and pointed (Fig. 14, *A* and *B*). These structures were displaced above McGregor's line but were considerably anterior to the foramen magnum, indicating that an atlanto-occipital subluxation had occurred. This was apparently followed by spontaneous atlanto-occipital fusion. There was bony ankylosis of the sacroiliac and virtually all of the posterior vertebral joints and extensive ossifications were present along the paravertebral ligaments.

Case x (L. R. No. 468244). A forty-nine year old male had the onset of spondylitis in 1935. Neck pain has been prominent, necessitating several courses of roentgen therapy (4,150 r between 1940 and 1947). He has had virtually complete spinal rigidity since 1950. The peripheral joints were never involved but temporomandibular joint motion was reduced. Comparative roentgenograms made in 1943 and 1960 showed atrophy of the odontoid and atlas with bony ankylosis of these structures to each other and to the basilar process (Fig. 15, *A* and *B*). He had erosions of the spinous processes of C6 and C7, calcification with reduction in the height of the cervical intervertebral disks and extensive bony ankylosis of the sacroiliac and posterior vertebral joints with paraspinal ossifications, characteristic of ankylosing spondylitis.



Fig. 9. Case v. Rheumatoid arthritis. Midline laminagram. "Stepladder" subluxations with reduction in the height of the intervertebral disks and erosions of the vertebral end-plates, posterior arches and odontoid process. The latter extends well above the palato-occipital line and the foramen magnum-clivus angle is 142°. The lower spinous processes appear "pointed" as does the basilar process. Note retro-odontoid soft tissue mass.

Discussion

The cervical spine is frequently affected in classic rheumatoid arthritis as well as in ankylosing spondylitis. Subluxations, especially atlanto-axial, often accompany such involvement but flexion views are generally required for the diagnosis. Pathologic laxity of the transverse ligament may account for minimal degrees of atlanto-odontoid separation. When this separation is severe, however, one may postulate

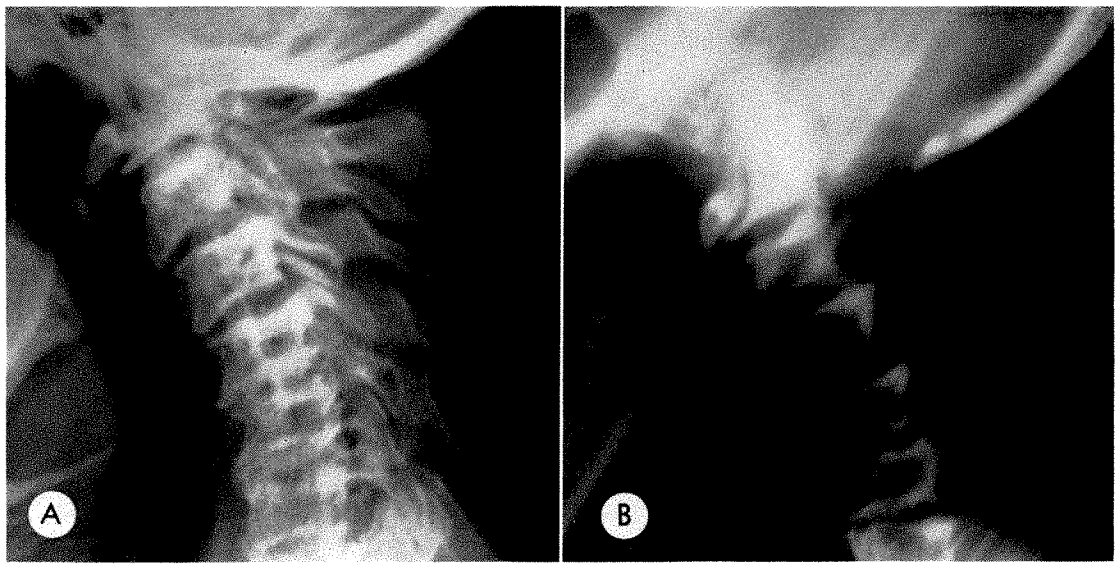


Fig. 10. Case VI. Rheumatoid arthritis. (*A*) Lateral view. Multiple cervical subluxations with severe erosions of the posterior arches and vertebral end-plates. The atlas has descended to the inferior margin of C2. (*B*) Midline laminagram. The body of C2 is sclerotic and eroded. The odontoid too is sclerotic and extends into the posterior fossa. The foramen magnum-clivus angle appears wide but the tip of the basilar process, as well as the occipital condyles and lateral masses have probably been eroded ("pseudo-basilar invagination").

rupture of this ligament; in such cases, the extent of forward dislocation of the atlas and skull is apparently variable and depends largely on the functional length of the alar ligaments.[44] Minimal widening of the atlanto-odontoid space in flexion, in the presence of odontoid erosion, does not necessarily signify an abnormality of the transverse ligament. The reduced diameter of the odontoid due to erosion may cause it to fit loosely in the ring formed by the anterior atlas and transverse ligament and may alone account for atlanto-odontoid instability. Cervical subluxation was less prevalent in ankylosing spondylitis but many had advanced bony fusion of the posterior articulations and extensive paraspinal ossifications. Such subluxations may occur more frequently during the active phase of this disease. It is possible, too, that neck pain is a less reliable indication of cervical subluxation in ankylosing spondylitis than in rheumatoid arthritis.

The occurrence of erosions of the odontoid process in these diseases is not surprising. In rheumatoid arthritis such lesions, as well as those in adjacent ligaments, are apparently identical to those in the peripheral joints (Fig. 4, *A* and *B*). One patient exhibited an erosion of the posterior aspect of the odontoid with an adjacent bone fragment resembling osteochondritis dissecans (Fig. 7). It is conceivable that the cartilaginous body, often found in the transverse ligament, may undergo osseous metaplasia, but in this case, the size and straight, smooth posterior aspect of the bony fragment suggested that it originated from the odontoid process. The lower cervical spinous processes either showed sharply marginated cortical erosions or, more often, their previously rounded ends became pointed, frequently appearing as though they had been amputated. The vertebral end plate erosions (Fig. 9), with reduction in the height of the adjacent intervertebral disks, at times resembled spinal tuberculosis. These occurred at multiple levels in 2 cases, at C2–C3 in one, and at C5–C6 in another. Thinning of the disks, without adjacent osteophytes or erosions, occurred in the upper half of the cervical spine in several patients in whom the disease began in adulthood.

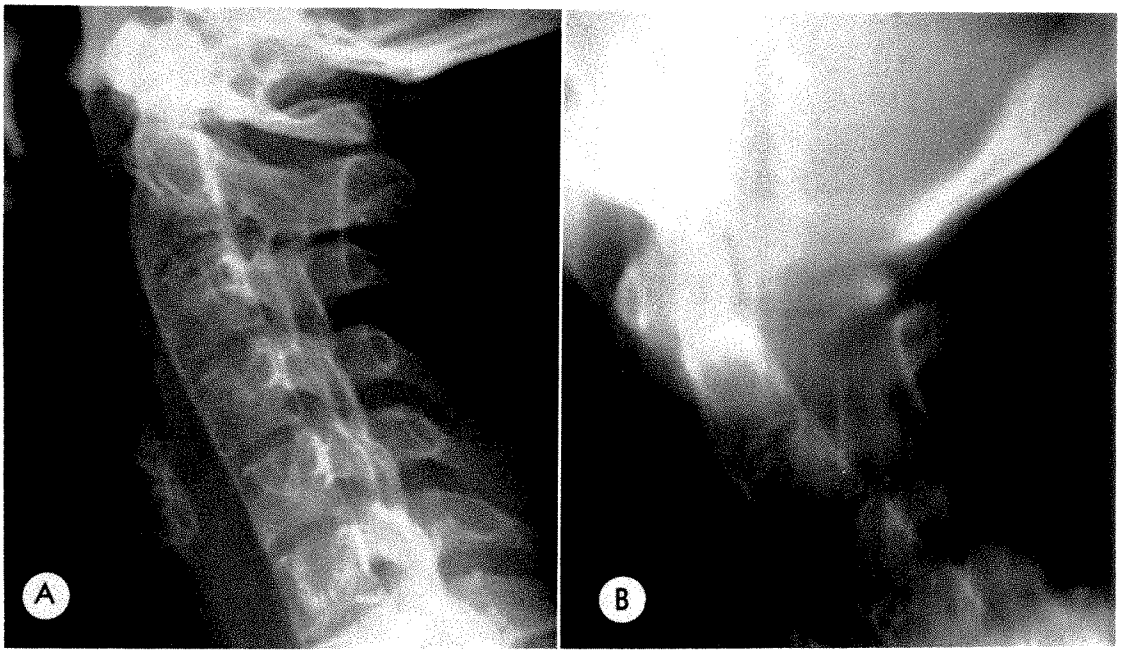


Fig. 11. Case VII. Ankylosing spondylitis. (*A*) 1954. Advanced cervical spondylitis. Normal alignment of the atlas. (*B*) 1960. Midline laminagram showing 5 mm. anterior displacement of the atlas and bone proliferation around the odontoid with fusion to the atlas in the dislocated position.

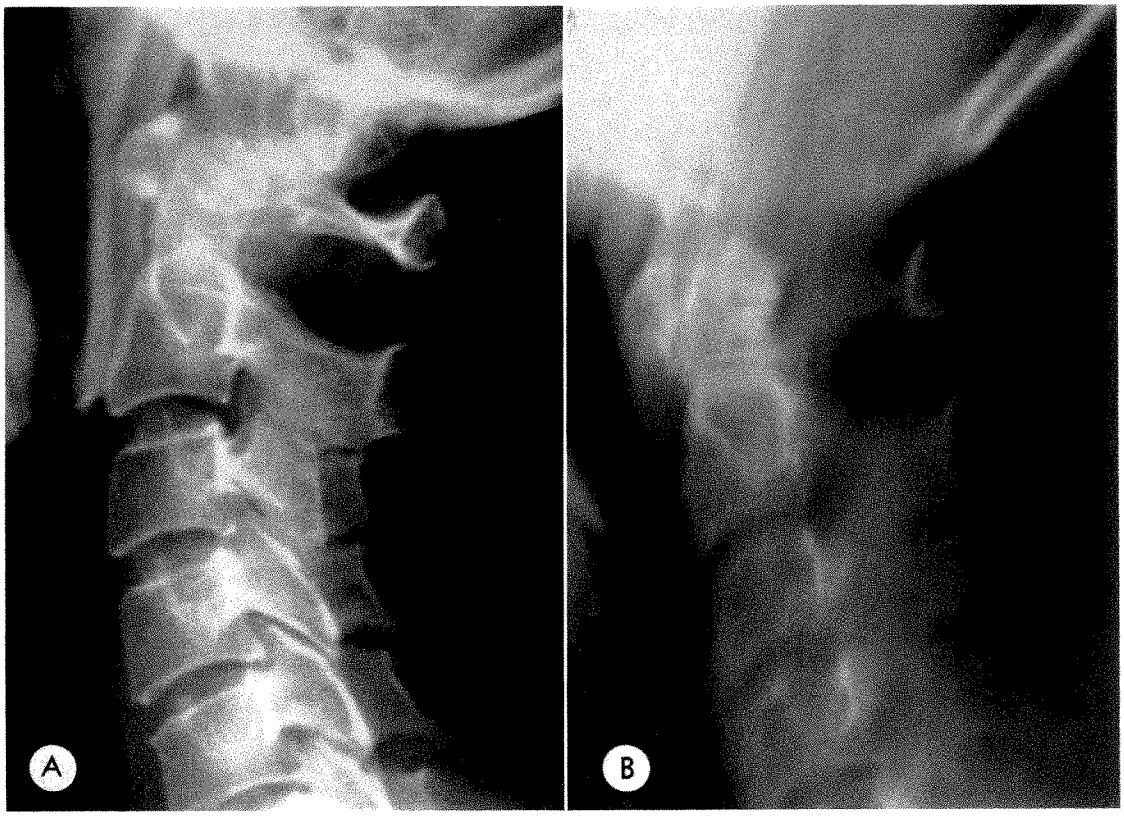


Fig. 12. Case VIII. Ankylosing spondylitis. (*A*) 1945. Normal odontoid. (*B*) 1960. Midline laminagram showing superficial erosions and reactive sclerosis of the odontoid with a mottled trabecular pattern.

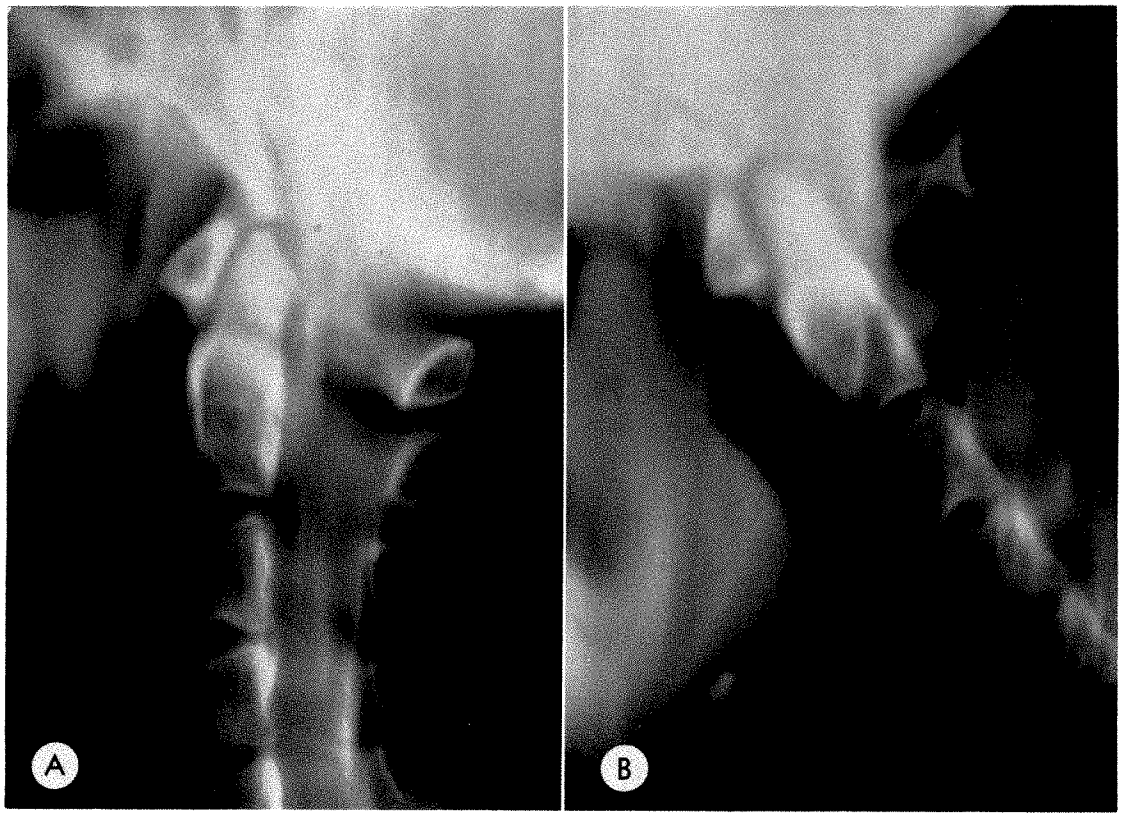


Fig. 13. Nineteen year old male. (*A*) Extension. (*B*) Flexion. Minimal atlanto-odontoid separation with flexion. The odontoid is sclerotic with superficial erosions. Neck and back pain were prominent and the sacroiliac joints showed characteristic changes of ankylosing spondylitis.

The surface erosions of the odontoid in ankylosing spondylitis appeared superficial and were usually associated with a reactive sclerosis, obliterating the normal trabecular pattern (Fig. 12, *A* and *B*; and 13). Such erosions with adjacent osteosclerosis have been described in the pelvic bones and vertebral bodies in this disease,[7,13,34] usually at points of ligamentous attachment, and presumably represent the same pathologic process as in the odontoid process. Romanus and Ydén[34] have referred to such lesions in the anterior corners of the vertebral bodies as "shining corners"; hence, the term "shining odontoid" is suggested. The atlas and odontoid became atrophic in 2 ankylosing spondylitis patients who had also developed either atlanto-occipital or atlanto-odontoid-occipital fusion (Case IX and X). These are not listed as "odontoid erosions" in Table II but were, in all probability, due to a subtle erosive process, giving the appearance of a "wearing down of the bone"[34] (Fig. 14 *B* and 15 *B*).

Occasionally, the sacroiliac joints, as well as the cervical spine, are involved in otherwise classic rheumatoid arthritis.[5,36] This appeared to be true in 4 cases in this series in which the sacroiliac joints were narrowed and showed superficial, sharply demarcated marginal irregularities, unattended by reactive sclerosis. Three of the 4 patients also had erosions of the ischial tuberosities. None had paraspinal ossifications (syndesmophytes).[34] These patients differed clinically as well as roentgenologically from those with typical ankylosing spondylitis[23] in that they had rheumatoid nodules and a strongly positive test for the rheumatoid factor, and none had significant limitation of back motion.

It was of interest to correlate the foramen

magnum-clivus angles with the odontoid-baseline distances in these patients and to compare them with values obtained in individuals of similar age distribution, not having rheumatoid disease (Fig. 16). In most cases these were either normal individuals or patients in whom there was a question of a skull fracture. Laminagrams were available in 12 instances. Care was taken to exclude those with proved intracranial pathology or generalized bone disease. There was a statistically significant departure from normal (t test) in the odontoid-baseline distance both in rheumatoid arthritis and ankylosing spondylitis. Upward displacement of the spine relative to the palato-occipital line may be caused by erosions of the cartilage and bone of the lateral atlanto-axial and atlanto-occipital joints and need not be associated with basilar invagination. However, apparently as the ondontoid-baseline distance increases, there is a tendency toward a wide foramen magnum-clivus angle. In rheumatoid arthritis, the foramen magnum-clivus angle differed significantly (t test, $P < .005$) from the control group. There was also a statistically significant difference in ankylosing spondylitis (t test, $.025 < P < .05$) but this was less marked than in rheumatoid arthritis. It is of interest to note that the mean values are not widely divergent (Table III). It appears that secondary basilar invagination can occur as a complication of rheumatoid arthritis and accounts for widening of the foramen magnum-clivus angle and vertical translation of the spine. However, in some patients the up-

TABLE III

MEAN VALUES FOR ODONTOID-BASELINE DISTANCE AND FORAMEN MAGNUM CLIVUS ANGLE

	Odontoid-Baseline Distance (mm.)	Foramen Magnum-Clivus Angle
"Normals"	0.38	121.3°
Rheumatoid Arthritis	3.62	125.6°
Ankylosing Spondylitis	3.03	123.4°

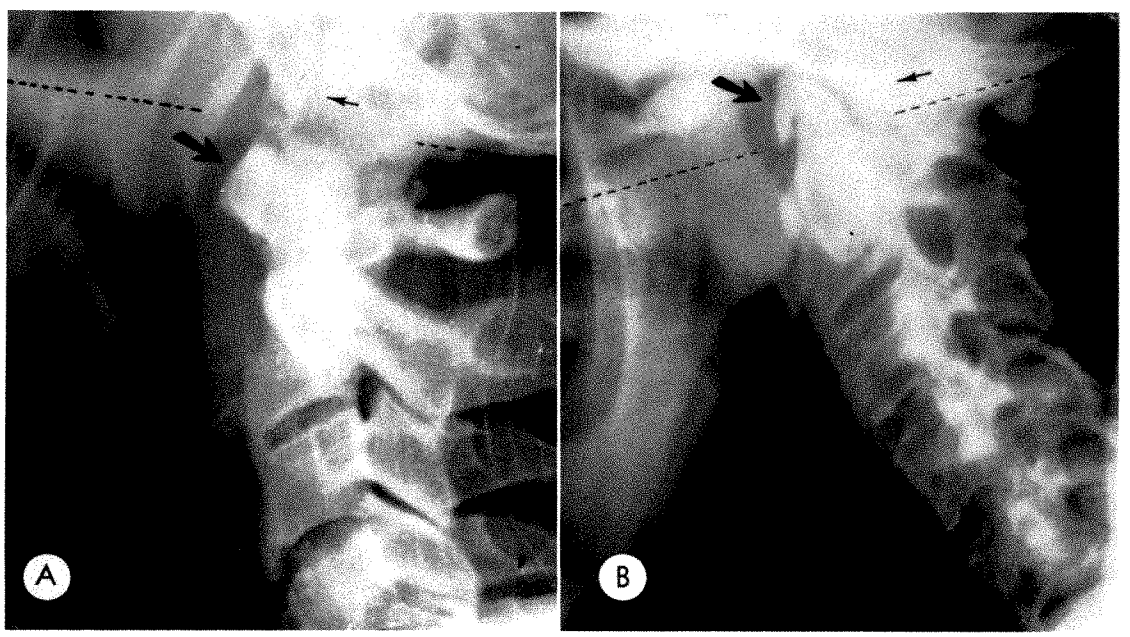


FIG. 14. Case IX. Ankylosing spondylitis. (*A*) 1952. (*B*) 1959. The atlas (large arrow) and odontoid have become atrophic, as though worn down, and displaced upward and anterior to the foramen magnum, indicating atlanto-occipital subluxation. Note tip of basilar process (small arrow) and McGregor's line. Atlanto-occipital fusion apparently followed. No basilar invagination. (Courtesy of *Arthritis and Rheumatism.*)

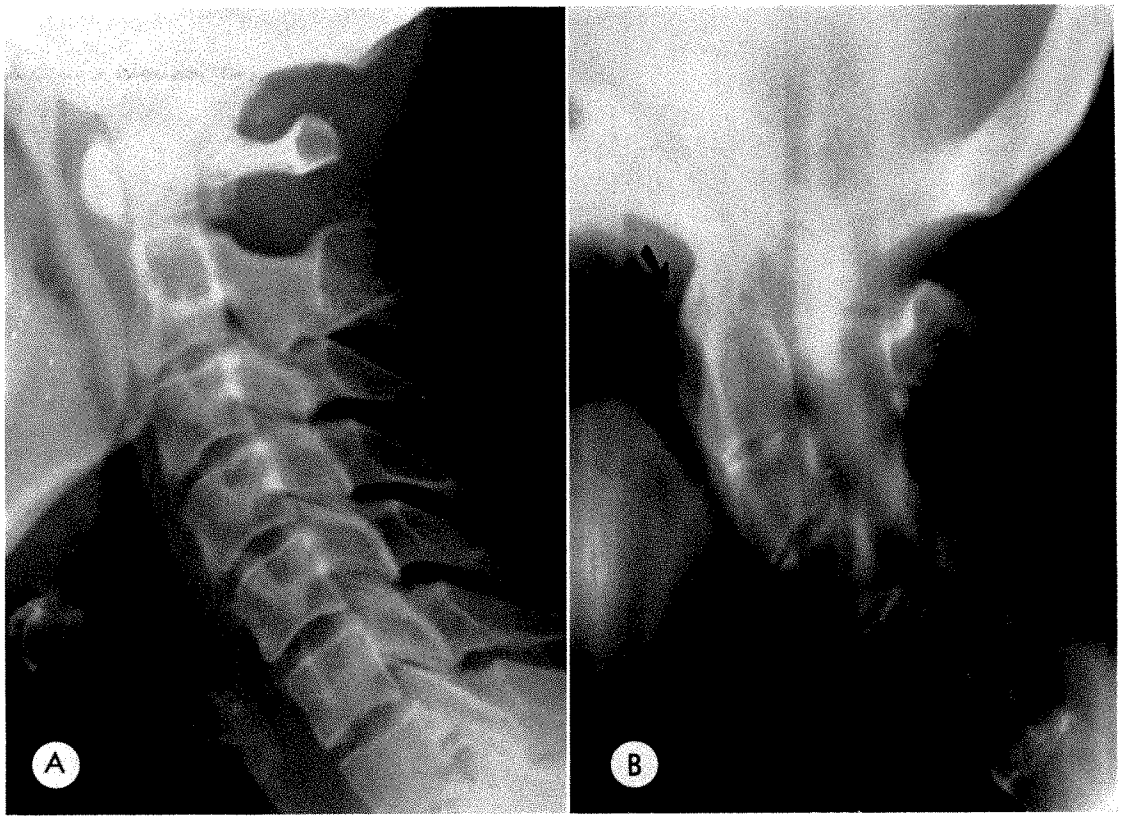


Fig. 15. Case x. Ankylosing spondylitis. (*A*) 1943. Normal atlas and odontoid. (*B*) Midline laminagram. The atlas (arrow) and odontoid have atrophied and are fused to one another and to the basilar process.

turning of the foramen magnum margins that is typical of basilar invagination is absent and yet the odontoid-baseline distance is markedly abnormal and the foramen magnum-clivus angle is wide. This was true in Case VI where the odontoid extended into the posterior fossa. One may postulate that in some patients not only are the lateral masses and occipital condyles eroded but the tip of the basilar process is likewise destroyed. Such erosions might allow the skull to descend on the spine, as it were, and would also give the appearance of a wide foramen magnum-clivus angle. This situation might properly be differentiated as a "pseudo-basilar invagination."

Laminagrams were indispensable for clearly demonstrating the odontoid lesions and delineating the precise odontoid-atlas relationship. They also showed the periodontoid soft tissues to advantage. In several patients with advanced bony lesions in this region, there was unusual prominence of these soft tissues, possibly due to inflammation. In one patient with basilar invagination, this may have represented brain tissue extending into the spinal canal (Case V).

The lack of significant neurologic manifestations was a perplexing finding. The slow development of these lesions allows an adaptation of the spinal cord, and the reduction in size of the odontoid might explain this discrepancy. However, in several instances the sagittal diameter of the spinal canal was less than the lower normal limits.[45,47]

Spontaneous atlanto-axial subluxation causing quadriplegia has been reported in ankylosing spondylitis,[21] and was also observed in rheumatoid arthritis with fatal spinal cord compression.[22] While muscular atrophy in rheumatoid disease is probably a peripheral effect in most cases, compres-

sion of the spinal cord at the foramen magnum or upper cervical spine may also produce such atrophy and loss of reflexes in the upper extremities. An anterior horn lesion may be due to compression of the anterior spinal artery. It is possible that in some cases significant neurologic manifestations are being overlooked or attributed to the peripheral effects of the disease, for in patients with advanced disease and crip-

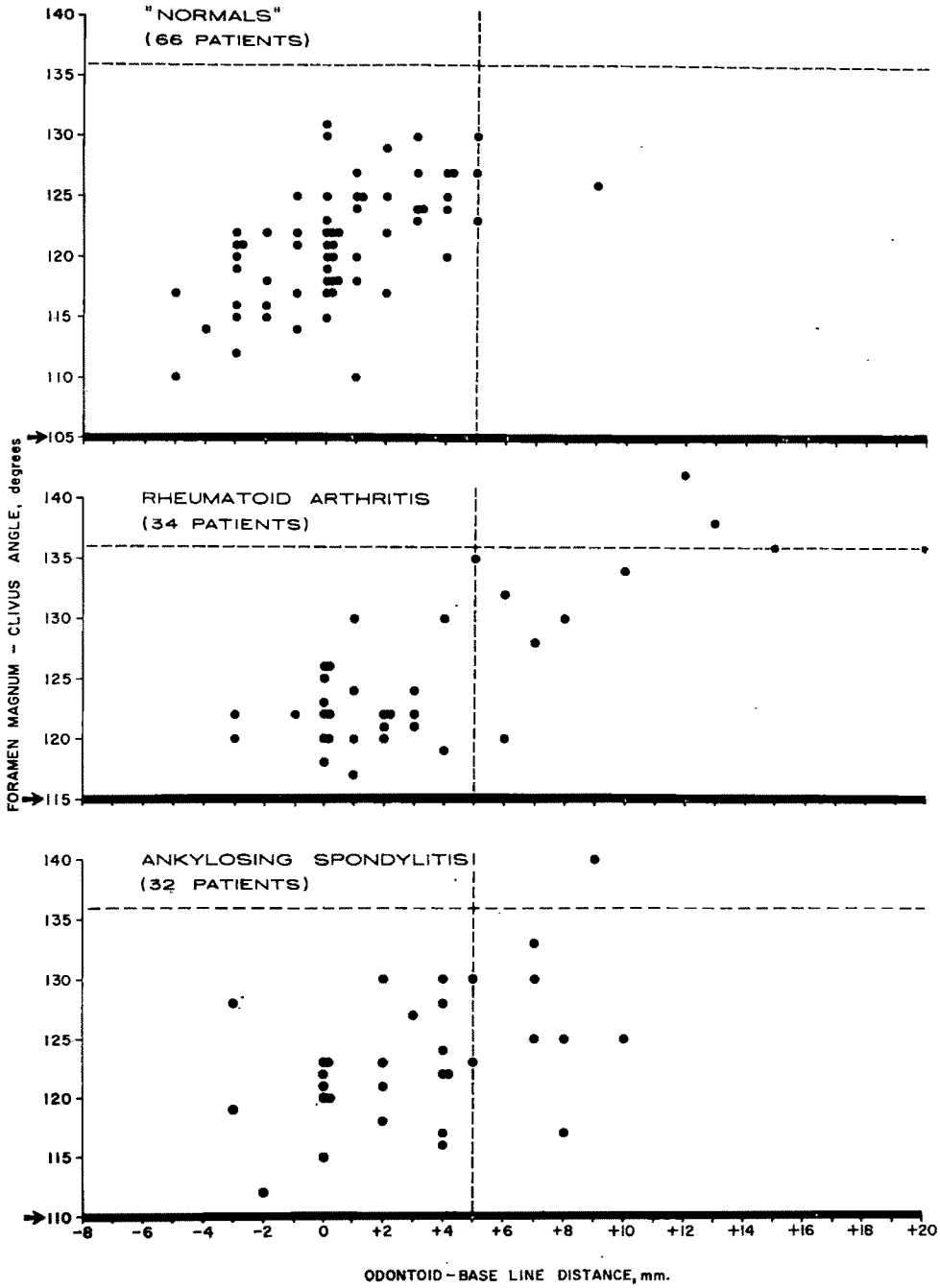


Fig. 16. Correlation of odontoid-baseline distance and foramen magnum-clivus angle in rheumatoid arthritis and ankylosing spondylitis and comparison with "normal" values. Interrupted lines represent the accepted upper normal limits.

pling deformities neurologic examination is often difficult. It is reasonable that chronic spinal cord compression or vertebral artery insufficiency[35] may develop in some instances. The latter may have been a significant factor in Case 1, precluding collateral blood flow in the presence of internal carotid artery occlusion. Reports of delayed myelopathy in traumatic atlanto-axial dislocation[1,43] and in congenital abnormalities around the foramen magnum[20] suggest that a similar asymptomatic interval may obtain in these patients.

Virtually all of the patients with rheumatoid arthritis received steroid therapy, but this may reflect the severity of the disease in these patients rather than a correlation with cervical subluxation. Atlanto-axial subluxation and odontoid erosion have been seen in patients who did not have steroids.[11,22] The prevalence of these potentially dangerous lesions in unselected patients with rheumatoid arthritis remains to be determined, and also whether their development can be correlated with prolonged steroid therapy, age of the patient, duration or severity of the disease, or any other factor.

DIFFERENTIAL DIAGNOSIS

Spontaneous bony fusion in the occipito-atlanto-axial joints of the type seen in ankylosing spondylitis may also occur in rheumatoid arthritis when the disease begins in childhood. This is consistent with the bony ankylosis that can occur in the posterior cervical joints in these cases. In one child with rheumatoid arthritis and chronic atlanto-axial dislocation of two years' duration, laminagrams disclosed periosteal new bone formation of the odontoid process. Children with nonspecific infections of the neck (*e.g.*, tonsillitis, mastoiditis) may develop spontaneous atlanto-axial dislocations.[15,39,41,44,46] This suggests that a specific rheumatoid lesion of the transverse ligament need not necessarily be postulated to explain atlanto-axial subluxation in juvenile rheumatoid arthritis. Congenital malformations,[20,27,29] such as primary basilar invagination, atlas assimilation, and dysplasia of the odontoid, must also be considered in the differential diagnosis. Septic arthritis, pyogenic[9] or tuberculous, rarely involves the upper cervical spine. Cervical subluxations may occur as a complication of degenerative spondylosis and osteoarthritis, but usually involve the lower cervical spine and are generally of minimal degree although at times they may be severe; however, involvement of the atlanto-odontoid joint is not apparently associated with atlanto-odontoid separation. Trauma to the completely ankylosed cervical spine in spondylitis may cause a fracture-dislocation. In these cases the spine fractures like a long bone, with the break usually occurring through what had formerly been the interspace.[19] Neurologic symptoms are probably more apt to occur in these instances than in the spontaneous subluxations in the nonankylosed spine. Multiple cervical subluxations, basilar invagination and severe destruction of the atlas, odontoid and possibly also the basilar process have been observed in lipoid dermatoarthritis in which there was severe involvement of the peripheral joints.[24]

SUMMARY

Rheumatoid arthritis and ankylosing spondylitis often involve the cervical spine, particularly the occipito-atlanto-axial joints. Nontraumatic cervical subluxation is frequent, especially in the atlanto-axial joints. Osseous erosions, particularly of the odontoid process, can often be demonstrated. Such surface erosions of the odontoid process in ankylosing spondylitis are often associated with a sclerosing osteitis. Basilar invagination may occur as a complication of rheumatoid arthritis, but in some cases the features are atypical and might more properly be called "pseudo-basilar invagination." These lesions of the cervical spine are potentially dangerous and often overlooked.

University Hospital
Department of Radiology
Ann Arbor, Michigan

The author is indebted to the staff of the Rackham Arthritis Research Unit, in particular to Dr. I. F. Duff, for his cooperation in making patients available and to Drs. F. J. Hodges, J. F. Holt, and W. M. Whitehouse for their continued interest and encouragement. The assistance given by Dr. R. D. Remington of the School of Public Health in the statistical analysis and by Dr. M. R. Abell of the Department of Pathology in evaluating the histologic material was greatly appreciated, as was the advice of Dr. R. T. Woodbourne of the Department of Anatomy.

REFERENCES

1. BACHS, A., BARRAQUER-BORDAS, L., BARRAQUER-FERRE, L., CANADELL, J. M., and MODOLELL, A. Delayed myelopathy following atlanto-axial dislocation by separated odontoid process. *Brain*, 1955, *78*, 537–553.
2. BOOGAARD, J. A. De indrukking der grandvlakte van den schedel door de vervelkolom, hare oorzaken en gevolgen. *Nederland Tijdschrift voor Geneeskunde*, 1865, *2*, 81–108.
3. BÖGER. Ein Fall von Malum suboccipitale rheumaticum. *Arch. f. orthop. u. Unfall-chir.*, 1905, *3*, 97–100.
4. BYSTROW, A. Assimilation des Atlas und Manifestation des Proatlas. *Ztschr. f. d. ges. Anat.* (Abt. 1), 1931, *95*, 210–242.
5. COHEN, A. S., MCNEIL, M., SHARP, J. T., SCHUBART, A. F., CALABRO, J. J., and CALKINS, E. Proceedings at Second Pan-American Congress on Rheumatic Diseases and Twenty-third Annual Meeting of American Rheumatism Association. *Bull. Rheum. Dis.*, 1959, *10*, 194.
6. COUDRAY, P. Sous-occipitale rheumatique. *Ann. de méd et chir. infant.*, 1900, *4*, 885–894.
7. CRUICKSHANK, B. Lesions of cartilaginous joints in ankylosing spondylitis. *J. Path. & Bact.*, 1956, *71*, 73–84.
8. CRUICKSHANK, B. Pathology of ankylosing spondylitis. *Bull. Rheum. Dis.*, 1960, *10*, 211–214.
9. FRANK, T. J. F. Osteomyelitis of odontoid process of axis. *Med. J. Australia*, 1944, *1*, 198–201.
10. GIBSON, H. J. Ankylosing spondylitis, etiology and pathology. *J. Fac. Radiologists*, 1957, *8*, 193–200.
11. GRAHAM, D. C. Personal communication.
12. GRAHAM, W. Is rheumatoid spondylitis a separate entity? *Arth. & Rheum.*, 1960, *3*, 88–90.
13. GUEST, C. M., and JACOBSON, H. G. Pelvic and extra pelvic osteopathy in rheumatoid spondylitis, clinical and roentgenographic study of 90 cases. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 760–768.
14. HADLEY, L. A. The Spine. Charles C Thomas, Publisher, Springfield, Ill., 1956, pp. 116–117.
15. HESS, J. H., BRONSTEIN, I. P., and ABELSON, S. M. Atlanto-axial dislocations; unassociated with trauma and secondary to inflammatory foci in neck. *Am. J. Dis. Child.*, 1935, *49*, 1137–1147.
16. JACKSON, H. Diagnosis of minimal atlanto-axial subluxation. *Brit. J. Radiol.*, 1950, *23*, 672–674.
17. JAKSCH, R. Un caso d'affezione poliartritica della colonna vertebrale cervicale. *Riforma Medica*, 1900, *1*, 581–582.
18. KORNBLUM, D., CLAYTON, M. L., and NASH, H. H. Nontraumatic cervical dislocations in rheumatoid spondylitis. *J.A.M.A.*, 1952, *149*, 431–435.
19. LEMMEN, L. J., and LAING, P. G. Fracture of cervical spine in patients with rheumatoid spondylitis. *J. Neurosurg.*, 1959, *16*, 542–550.
20. LIST, C. F. Neurologic syndromes accompanying developmental anomalies of occipital bone, atlas and axis. *Arch. Neurol. & Psychiat.*, 1941, *45*, 577–616.
21. MARGULIES, M. E., KATZ, I., and ROSENBERG, M. Spontaneous dislocation of atlanto-axial joint in rheumatoid spondylitis; recovery from quadriplegia following surgical decompression. *Neurology*, 1955, *5*, 290–294.
22. MARTEL, W., and PAGE, J. Cervical vertebral erosions in rheumatoid arthritis and ankylosing spondylitis. *Arth. & Rheum.*, 1960, *3*, 546–556.
23. MARTEL, W., and DUFF, I. F. Pelvo-spondylitis in rheumatoid arthritis. Presented at Interim Session of American Rheumatism Association, 1960.
24. MARTEL, W., ABELL, M. R., and DUFF, I. F. Cervical spine involvement in lipoid dermatoarthritis. *Radiology*. In Press.
25. MARTIN, R. C. Atlas-axis dislocation following cervical infection. *J.A.M.A.*, 1942, *118*, 874–875.
26. MCGREGOR, M. Significance of certain measurements of skull in diagnosis of basilar impression. *Brit. J. Radiol.*, 1948, *21*, 171–181.
27. MCRAE, D. L. Significance of abnormalities of cervical spine; Caldwell lecture, 1959. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *8*, 3–25.
28. MCRAE, D. L. Bony abnormalities in region of foramen magnum: correlation of anatomic and neurologic findings. *Acta radiol.*, 1953, *40*, 335–355.
29. PEYTON, W. T., and PETERSON, H. O. Congenital deformities in region of foramen magnum: basilar impression. *Radiology*, 1942, *38*, 131–144.

30. Poirier, P. Traité d'anatomie humaine. Cited by Trolard.[42] Paris, 1893.
31. Pratt, T. L. C. Spontaneous dislocation of atlanto-axial articulation occurring in ankylosing spondylitis and rheumatoid arthritis. *J. Fac. Radiologists*, 1959, *10*, 40–43.
32. Purser, D. W., and Sharp, J. Spontaneous atlanto-axial dislocation, complication of spondylitis. *J. Bone & Joint Surg.*, 1957, *39-B*, 582.
33. Rand, C. W. The Neurosurgical Patient: His Problems of Diagnosis and Care. Charles C Thomas, Publisher, Springfield, Ill., 1944, p. 47.
34. Romanus, R., and Yden, S. Pelvo-spondylitis Ossificans. Munksgaard, Copenhagen, 1955.
35. Schneider, R. C., and Crosby, E. C. Vascular insufficiency of brain stem and spinal cord in spinal trauma. *Neurology*, 1959, *9*, 643–656.
36. Sharp, J. Differential diagnosis of ankylosing spondylitis. *Brit. Med. J.*, 1957, *1*, 975–978.
37. Sharp, J., Purser, D. W., and Lawrence, J. S. Rheumatoid arthritis of cervical spine in adult. *Ann. Rheum. Dis.*, 1958, *17*, 303–313.
38. Stammers, F. A. R., and Frazer, P. Spontaneous dislocation of atlas. *Lancet*, 1933, *225*, 1203–1205.
39. Stein, F., Bloch, H., and Kenin, A. Non traumatic subluxation of atlanto-axial articulation; report of case. *J.A.M.A.*, 1953, *152*, 131–132.
40. Steinbrocker, O., Traeger, C. H., and Batterman, R. C. Therapeutic criteria in rheumatoid arthritis. *J.A.M.A.*, 1949, *140*, 959–662.
41. Sullivan, A. W. Subluxation of atlanto-axial joint: sequel to inflammatory processes of neck. *J. Pediat.*, 1949, *35*, 451–464.
42. Trolard. Les articulations de la tête avec la colonne vertébrale. *J. d'Anatomie*, 1897, *33*, 105–128.
43. Wade, L. J. Pseudo-platybasia: rupture of transverse ligament of axis with displacement of odontoid process and compression of cervical cord. *J. Bone & Joint Surg.*, 1941, *23*, 37–43.
44. Werne, S. Studies in spontaneous atlas dislocation. *Acta orthop. scandinav.*, 1957, Suppl. 23.
45. Wholey, M. H., Bruwer, A. J., and Baker, H. L. Lateral roentgenogram of neck, with comments on atlanto-odontoid-basion relationship. *Radiology*, 1958, *71*, 350–356.
46. Wilson, M. J., Michele, A. A., and Jacobson, E. W. Spontaneous dislocation of atlanto-axial articulation, including report of case with quadriplegia. *J. Bone & Joint Surg.*, 1940, *22*, 698–707.
47. Wolf, B. S., Khilnani, M., and Malis, L. Sagittal diameter of bony cervical spinal canal and its significance in cervical spondylosis. *J. Mt. Sinai Hosp*, 1956, *23*, 283–292.

VOL. 86, No. 2

DESTRUCTIVE LESIONS OF THE VERTEBRAL BODIES IN RHEUMATOID DISEASE*

By WILLIAM B. SEAMAN, M.D., *and* JOSEPHINE WELLS, M.D.
NEW YORK, NEW YORK

DURING recent years there have been occasional reports of destructive lesions of the vertebral bodies occurring in patients with rheumatoid disease.[3,10,19,20,25] These osteopathic lesions of the vertebrae constitute a confusing but perhaps significant feature of the disease.

The first description of a locally destructive lesion involving the upper and lower surfaces of the vertebral bodies was in an article by Andersson[1] on 2 patients with Marie-Strümpell arthritis. Edström[7] in 1940 reported several patients with erosion of the upper and lower corners of the upper lumbar and lower dorsal spine. One patient with a destructive area in the lower anterior portion of the twelfth dorsal vertebra was thought to have a tuberculous spondylitis because of the roentgenographic appearance. This diagnosis was never confirmed and the author concluded that it was a manifestation of rheumatoid spondylitis. Forestier *et al.*[8] described 2 cases with destructive vertebral lesions in a series of 200 cases of Marie-Strümpell arthritis. Roentgenographically, the lesions resembled those of tuberculosis but this disease was never proved. Guest and Jacobson,[10] in discussing osteopathic vertebral lesions occurring in 7 patients with rheumatoid spondylitis, illustrated one with a destructive lesion "involving the adjacent surfaces of the bodies of the eleventh and twelfth thoracic vertebra with peripheral eburnation and disk narrowing (not unlike tuberculous spondylitis)." In 1952, Baggenstoss, Bickel and Ward[3] reported 3 cases of rheumatoid spondylitis with similar destructive lesions of the vertebral bodies. These authors also described a patient with peripheral rheumatoid arthritis and destructive lesions of two vertebral bodies which, at autopsy, proved to be granulomatous, similar histologically to granulomatous nodules found in the synovia, heart and subcutaneous tissues. A more recent report by Wholey, Pugh and Bickel[25] from the same institution described 10 patients with similar roentgenographic lesions.

Romanus and Ydén,[20] after a careful clinical and roentgenographic analysis of 250 cases of rheumatoid spondylitis, concluded that destructive vertebral lesions were not merely sporadic phenomena occurring in occasional cases of rheumatoid spondylitis but could be found in the majority of cases, although they did not state precisely the incidence in their series. According to these authors, the destructive phase is followed relatively soon by a reparative phase that may result in complete healing with no trace of the spondylitic lesions; this may be the reason why it has not been commonly observed. Since roentgenograms are taken at infrequent intervals, these intermediary phases may be undetected.

Romanus and Ydén[20] describe the earliest change as a loss of bony cortex in a small area near the upper and lower anterior corners of the vertebral body which may be accompanied by a varying degree of sclerosis in the adjacent bone. As this progresses, the anterior borders of the vertebral bodies lose their normal convexity and this is responsible for the so-called square vertebrae characteristic of Marie-Strümpell arthritis. These authors feel that only in exceptional cases is this "squaring" due to bone deposition on the central third of the anterior portion of the vertebral body. In several of their patients, such destructive lesions of the vertebral bodies were the earliest roentgenographically demonstrable

* From the Department of Radiology, College of Physicians and Surgeons, Columbia University and Presbyterian Hospital in the City of New York.

TABLE I

Case No.	Patient	Age at Time of Destructive Lesion	Sex	Type of Arthritis	Duration of Arthritis Preceding Destructive Lesion (yr.)	Site of Destruction	Cortisone Treatment
I	D.L. 147-64-36	44	F	? clinically Marie-Strümpell; roentgenographically Marie-Strümpell	1	Mid-dorsal and upper lumbar	No
II	M.K. 30-94-57	40	M	Marie-Strümpell	17	L2, L3	No
III	H.W. 06-67-39	34	F	Marie-Strümpell	7	L1, L2	Yes (8 yr.)
IV	I.P. 08-89-67	55	F	Peripheral rheumatoid arthritis	9	T8, T9	Yes
V	N.B. 03-75-84	48	F	Peripheral rheumatoid arthritis	11	C6, C7	Yes (9 yr.)
VI	F.D. 79-94-59	36	M	Marie-Strümpell	18	T5, T6	Yes
VII	H.D. 01-85-79	29	M	Marie-Strümpell	9	L3, L4	Yes
VIII	H.E. 11-94-82	53	M	Marie-Strümpell	12	T7, T8, T11, T12	Yes (2 yr.)
IX	H.R. 147-48-29	47	M	? clinically Marie-Strümpell; roentgenographically Marie-Strümpell	3	L2, L3, L4	No
X	H.K. 04-76-92	27	M	Marie-Strümpell	10	L2, L3	No
XI	S.C. 96-31-33	38	M	Marie-Strümpell	15	T10, T11, T12	Yes
XII	R.A. 17-40-29	39	F	Marie-Strümpell	8	L2, L3, L5	Yes
XIII	M.H. 66-63-75	58	F	Marie-Strümpell	24	T10, T11	No

sign of spinal involvement. Although it always occurred after involvement of the sacroiliac joints, it was frequently a helpful diagnostic sign. There seems to be a predilection for the vertebral bodies at the dorsolumbar and lumbosacral junction.

Our experience during the last few years with several patients who had similar destructive lesions of the vertebral bodies prompted a review of the roentgenograms of 110 patients with Marie-Strümpell arthritis. This survey disclosed 11 patients

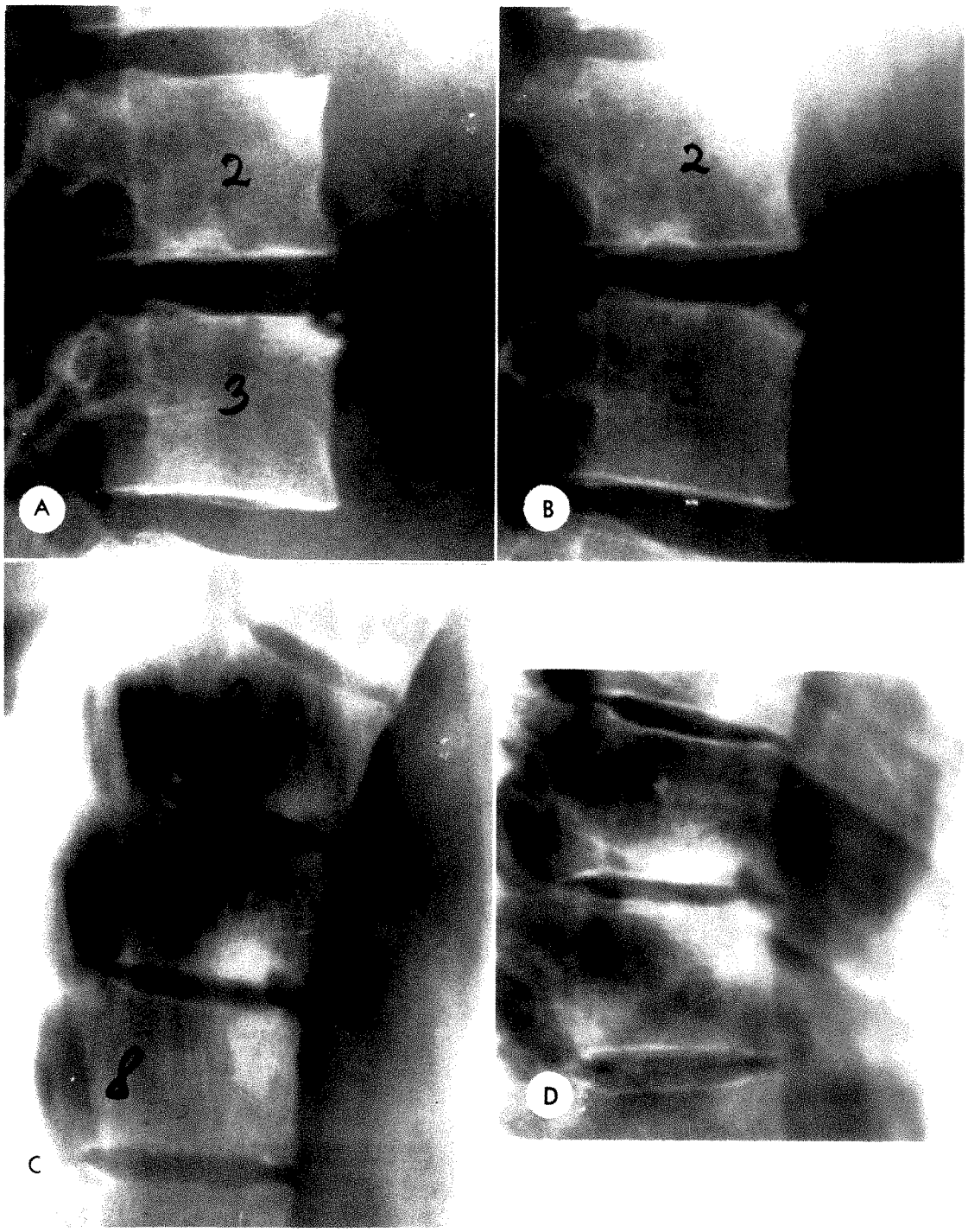


FIG. 1. D. L. (U.#147-64-36), a forty-eight year old female with rheumatoid spondylitis and a four year history of lower dorsal and upper lumbar pain. No steroid therapy. (*A*) Lateral view of the lumbar spine showing erosion of the upper anterior margin of L_3 with adjacent sclerosis. Similar but less marked changes are present in the upper anterior margin of L_2. (*B*) Four years later there has been only slight progression. (*C*) Lateral view of the dorsal spine showing an irregular defect with adjacent sclerosis in the lower anterior margin of T_7. Similar but less marked defects are present in T_5 and T6. (*D*) A detail view of T_7 four years later demonstrating a very slow rate of progression.

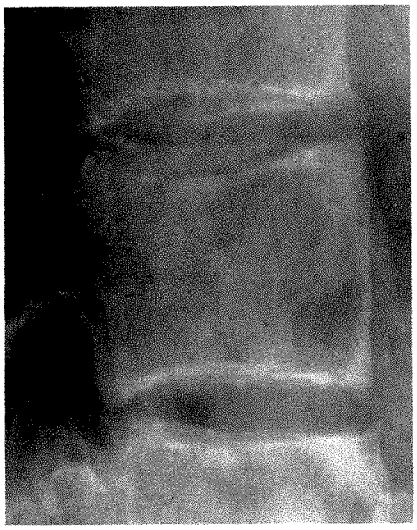


FIG. 2. M. K. (U.#30-94-57), a forty year old male with a seventeen year history of rheumatoid spondylitis. This lateral view of the lumbar spine reveals sclerosis of the anterior corners of L2 and the adjacent corners of L1 and L3. In addition, erosion of the upper anterior corners of L2 and L3 has resulted in the formation of pointed spurs. No steroid therapy.

with this type of process and, in addition, we found 2 cases in patients with peripheral rheumatoid arthritis (Table 1). There were several others who had definite but minimal sclerosis and erosion; they were not included.

Although we were unable to substantiate the statement of Romanus and Ydén that these lesions occur in the majority of cases, we were impressed by the long period of time that intervened between roentgenographic studies in many patients; so they may be correct in their assumption that this stage of the disease, which may be transient, is overlooked.

ROENTGENOGRAPHIC APPEARANCE

The roentgenographic lesions are of two main types which initially seem quite similar but differ in that one progresses to involve the entire extent of the cortical plates of the adjacent vertebral bodies while the second, conceivably an abortive manifestation, seems to remain localized to the anterior corner of a vertebral body (Fig. 1, *A–D*). They both begin as a tiny focus of destruction near the upper or lower anterior corner of the vertebral body associated with varying degrees of sclerosis of the adjacent bone. This may result in sharpening of the corner and a loss of its normal forward convexity so that it projects as a pointed spur (Fig. 2). This process, according to Romanus and Ydén, is responsible for the square vertebra characteristic of rheumatoid spondylitis. This type of destruction was infrequent in our material. Some patients exhibit a small area of destruction without any associated bony sclerosis (Fig. 6). In others the erosion is minimal and the lesion is characterized chiefly by sclerosis (Fig. 7, *A* and *B*).

In other instances, the initial lytic area spreads and the entire surface of the adjacent cortical plates is destroyed. After this occurs, the process seems to remain stationary and little or no further progress is recognizable roentgenographically (Fig. 3, *A-C*).

Two patients of this group, both females, were classified as having peripheral rheumatoid arthritis rather than rheumatoid spondylitis, and they exhibited some differences in the roentgenographic appearance and course of the disease. In these patients, a small initial lytic focus was not observed and the first demonstrable lesion was extensive, involving all of the cortical plates of both adjacent vertebral bodies with rapid complete destruction of the intervertebral disk (Fig. 4, *A–C;* and 5, *A–D*). The lesion then progressed to more or less complete healing so that the end stage resembled the healed stage of tuberculous spondylitis with no remaining trace of the intervertebral disk or adjacent body cortical plate.

ATLANTOAXIAL SUBLUXATION

This unusual complication occurred in a patient with peripheral rheumatoid arthritis who also had a destructive process involving the interspace between the fifth and sixth cervical vertebrae (Fig. 5, *C* and *D*). Laminagraphic studies also revealed evidence of bone erosion of the surface of the odontoid and the anterior arch of the

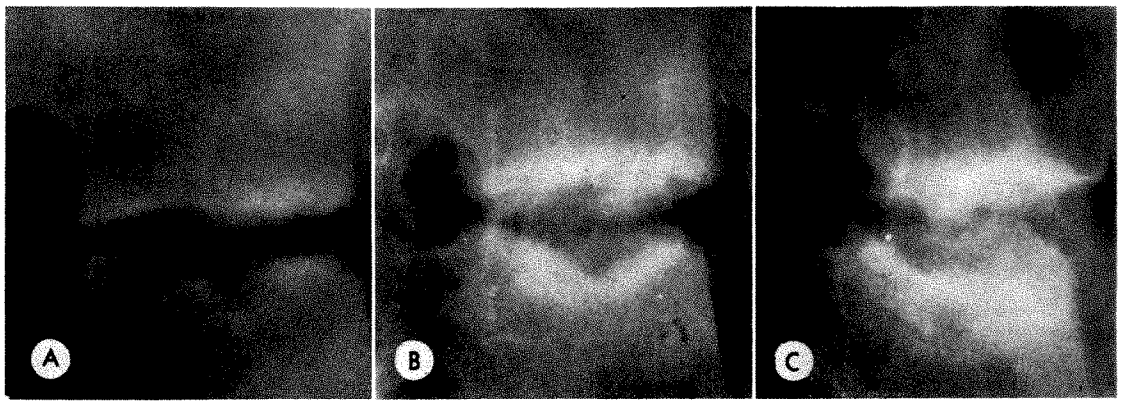


FIG. 3. H. W. (U.#06-67-39), a forty-one year old female with a sixteen year history (in 1960) of rheumatoid spondylitis. (*A*) 1951: A lateral view of L1 and L2 after two years of steroid therapy revealing minimal erosion and sclerosis of the anterior portions of the cortical plates of the adjacent vertebral bodies. (*B*) Eight months later the process has spread to involve the entire intervertebral disk and adjacent vertebral bodies. (*C*) Almost three years after *B* there is only minimal progression. The clinical condition was essentially the same and she has continued to receive cortisone therapy.

first cervical vertebra. Although she complained of severe neck pain, there was no clinical evidence of cord compression. She was treated with a neck brace and has been doing well now for the four years since the discovery of the subluxation. A second patient, not included in this series, with peripheral rheumatoid arthritis and less severe atlantoaxial subluxation, also complained of neck pain and had no neurologic findings. A third patient with rheumatoid spondylitis, also not included, showed evidence of C1–C2 subluxation but no destruction was seen.

Although there are numerous reports describing atlantoaxial dislocation due to acute cervical or nasopharyngeal inflammations,[6] there are relatively few recorded

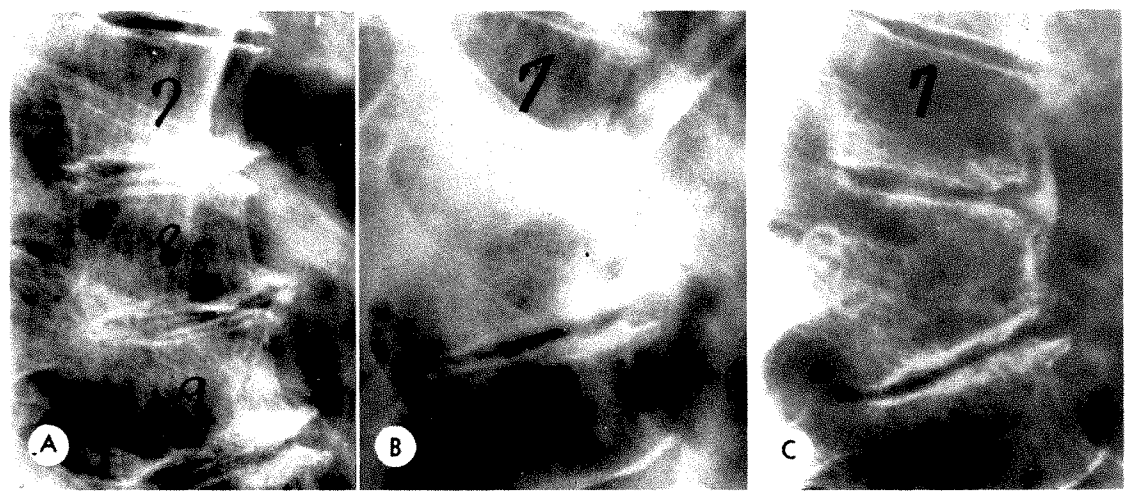


FIG. 4. I. P. (U.#08-89-67), a fifty-eight year old female with a twelve year history (in 1960) of peripheral rheumatoid arthritis. She was treated with ACTH for one month in 1952 and with meticorten from February 7, 1957 to February 23, 1957. (*A*) A lateral view of the thoracic spine, taken in February, 1957 because of severe lower dorsal pain, was interpreted as showing osteoarthritis. (*B*) Seven weeks later there is complete destruction of the intervertebral disk between T8 and T9. Tuberculin test was negative (1:1,000); streptococcal agglutination test was positive. No antibiotic therapy was given. (*C*) A roentgen study three years later shows a healed state with fusion of T8 and T9 and complete disappearance of the intervertebral disk.

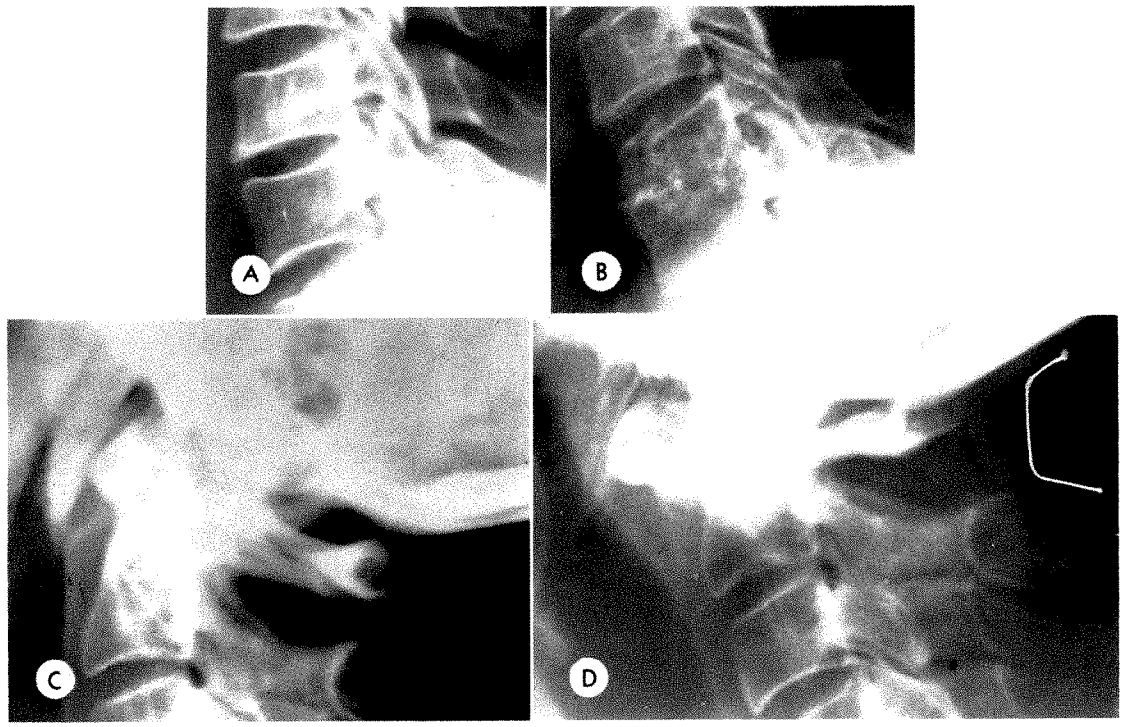


FIG. 5. N. B. (U.#03-75-64), a forty-eight year old female with an eleven year history (in 1956) of peripheral arthritis. She had been treated intermittently with cortisone for seven years. (*A*) A lateral view of C6 and C7 taken in 1954 and interpreted as showing a narrow disk space. (*B*) Lateral roentgenogram of C6 and C7 taken three years later because of severe neck pain shows almost complete destruction of the intervertebral disk and the cortical plates of adjacent vertebral bodies. (*C*) Roentgenogram showing the normal appearance of the atlantoaxial joint in 1954. (*D*) In 1957 there was marked posterior and upward displacement of the odontoid and the body of C2.

in patients with rheumatoid spondylitis or rheumatoid arthritis. Purser and Sharp[17] stated that they had seen 17 examples of atlantoaxial dislocation occurring in patients with ankylosing spondylitis and 23 instances in patients with rheumatoid arthritis. They suggested that many cases may be overlooked by failing to take lateral roentgenograms of the cervical spine in flexion. According to Watson-Jones,[23] the hyperemia accompanying inflammations results in softening of the atlas. This may be sufficient to allow detachment of the transverse ligament, which is mainly responsible for maintaining normal relationships between the first and second cervical vertebrae. Margulies *et al.*[13] described atlantoaxial dislocation producing a Brown-Séquard syndrome in a patient with rheumatoid arthritis. Microscopic examination of bone removed at the time of surgery revealed replacement of marrow spaces with fibro-collagenous tissue containing a dense inflammatory infiltrate. Rand[19] also reported a patient who had ankylosing spondylitis with a Brown-Séquard syndrome due to atlantoaxial dislocation, but the association of the two conditions was not recognized. Although the onset of neurologic symptoms may be abrupt and even sudden death may occur, in 85 to 90 per cent the only symptom is painful torticollis.[23] Pratt[16] in 1959 reported 3 more examples and was able to collect 53 cases from the literature (including the 40 mentioned by Purser and Sharp). More recently, Martel[14] described 29 additional cases and he also emphasized that they are usually overlooked unless lateral views of the cervical spine in flexion are obtained.

DISCUSSION

The 11 cases of Marie-Strümpell arthritis with destructive lesions of the spine have certain features in common. The destruction begins anteriorly and remains relatively stationary or it may progress to involve the entire articular surface of the vertebra. The disk space is not always narrowed. In all of these cases there was evidence of disease of the sacroiliac joints, and the patients had had symptoms for one year or more. Four of 11 patients were females, which is a higher incidence than usually is encountered in rheumatoid spondylitis, but the series is too small to be significant. Most of the patients complained of moderate to severe pain in the region of the destructive process, but a few patients had no symptoms referable to the site of the lesion.

The roentgenographic appearance may be indistinguishable from that produced by tuberculosis, Staphylococcus, typhoid, parathyphoid or Brucella infection of the vertebra. There are a few minor differences in that, with bacterial infections, narrowing of the intervertebral disk usually precedes roentgenographic evidence of bone destruction, while in rheumatoid disease bone erosion is frequently demonstrable without disk narrowing and occasionally even with disk widening (Fig. 3, *A–C*). In some cases disk narrowing develops later (Fig. 7, *A* and *B;* 8, *A* and *B*). Paravertebral masses are frequently associated with bacterial osteomyelitis of the spine, while they are rare in rheumatoid disease. The rate of destruction of the intervertebral disk classically is rapid in pyogenic osteomyelitis and relatively slow in tuberculous infection,[15] but in both it progresses more rapidly than in the majority of our patients. Wholey *et al.*[25] also stated that the lesions in their 10 patients failed to show the progression that would be expected of other infectious or granulomatous processes. Once the entire intervertebral space and cortical plates of adjacent vertebral bodies were involved, the process seemed to remain static, except for the 2 patients in whom spontaneous healing occurred. Tuberculous skin testing was done in only 1 patient in this series (Case IV, Table 1; Fig. 4) and that was negative at 1:1,000 dilution. No patient received specific antituberculous therapy.



FIG. 6. F. D. (U.#79-94-59), a thirty-six year old male with an eighteen year history of rheumatoid spondylitis. On this roentgenogram made in 1954 note a destructive lesion involving the intervertebral disk between T5 and T6 and destruction of the adjacent bone anteriorly. The patient had no back symptoms at the time of this study. Cortisone therapy had been given two and four years earlier, for ten days each time, because of uveitis.

The role of steroid therapy in the pathogenesis is difficult to assess. That it is not the sole causative factor is evidenced by the 5 patients in this series who were not treated with this type of drug. There are also reports of several cases in the literature which were not treated with steroids.[19] Of the 8 patients who did receive steroid therapy, 3 had received only one short course (two to three weeks) of cortisone or ACTH two or three years prior to the discovery of the osteolytic spinal lesion. On the other

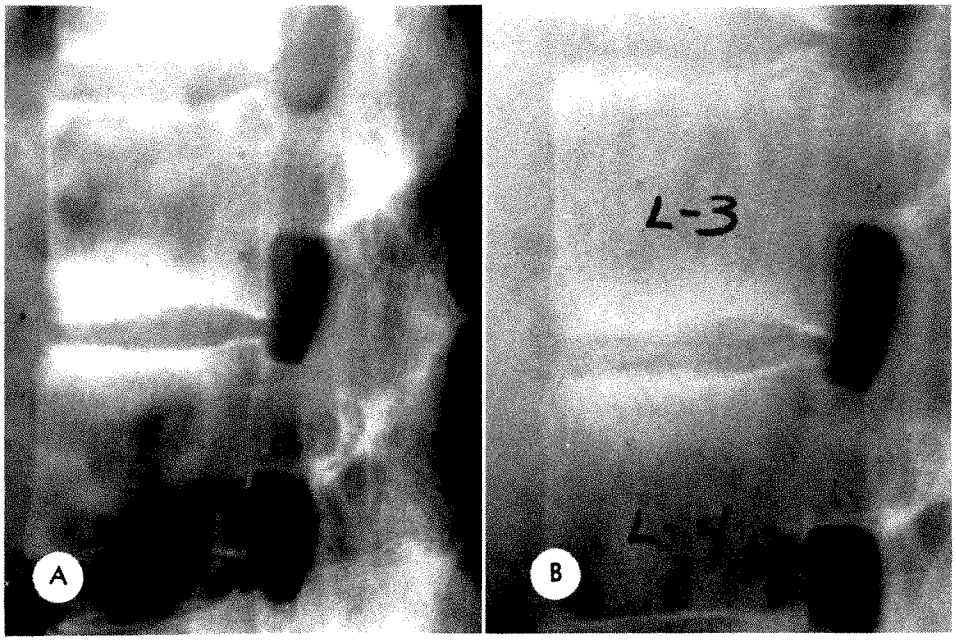


Fig. 7. H. D. (U.#01-85-79), a twenty-nine year old male with a nine year history of rheumatoid spondylitis. A ten week course of cortisone had been given in 1950. (*A*) A lateral view of the lumbar spine in 1952 showing sclerosis of the L_3 and L_4 vertebral bodies adjacent to the intervertebral disk. Note the minimal erosion of the anteroinferior corner of L_3 and the anterosuperior border of L_4. (*B*) Two years later the vertebral margins are smoother and the sclerosis has diminished. This process may be the pathogenesis of the square vertebra seen in rheumatoid spondylitis.

hand, 3 patients were on cortisone continuously for two years, eight years and nine years, respectively. One possible etiologic mechanism is the potentiation of any type of infection, particularly tuberculosis, because of suppression of the body's immune mechanism by the steroid. Steiner[22] has reported experiences with patients on prolonged cortisone therapy in whom at autopsy multiple infective lesions of the spine were demonstrated. During life, these patients were asymptomatic and roentgenographically negative. Most of our cases which had extensive destruction were treated with steroids[3] with the exception of Cases 1 and 11. On the other hand, 2 patients were continued on cortisone after the spinal lesion was demonstrable without any clinical or roentgenographic progression of the disease.

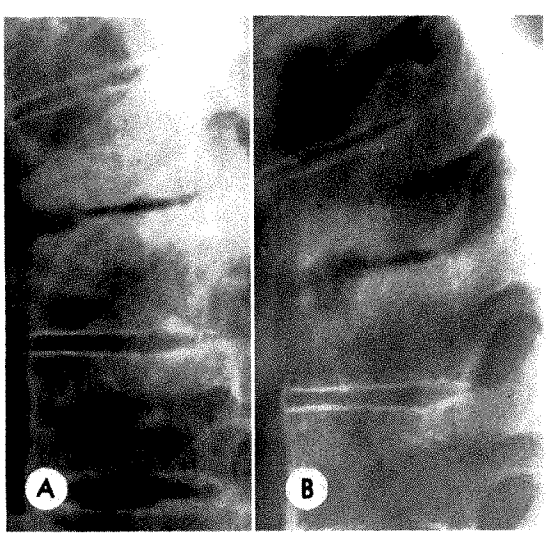


Fig. 8. H. E. (U.#11-94-82), a fifty-three year old male with a history of rheumatoid spondylitis for twelve years. He was treated with cortisone for two years. (*A*) A lateral view of the thoracic spine demonstrating a destructive lesion involving the 7th and 8th dorsal vertebral bodies adjacent to the intervertebral disk. (*B*) One year later the destructive process had extended to involve the entire extent of the intervertebral disk and the adjacent cortical plates.

The destruction and disappearance of the femoral head that may occur during steroid therapy[2,11,18] suggest that a similar phenomenon could occur in the spine. Murray[15] described a patient with purpura who was treated with 40 mg. of prednisolone daily for six days and who developed a similar destructive lesion of the anterior portion of the intervertebral disk and adjacent bone. Although the presumptive diagnosis was tuberculosis and the patient promptly healed on antituberculous therapy, bacteriologic verification was never obtained. Ansell[2] reported a patient with acute pancreatitis and hepatitis who developed a destructive vertebral lesion after thirteen days of treatment with ACTH. This was also assumed to be tuberculosis and was treated accordingly, although the etiologic agent was never discovered. It is possible that most of these lesions may represent rheumatoid involvement of the spine with granulation tissue similar to that in the autopsied patient reported by Baggenstoss *et al.*[3]

CONCLUSION

1. Thirteen patients, 11 with rheumatoid spondylitis and 2 with peripheral rheumatoid arthritis, with destructive lesions of the vertebral bodies are reported.

2. The roentgenographic appearance of the lesion simulates bacterial osteomyelitis of the vertebra but the clinical and roentgenographic course fails to show the progression usually seen in bacterial infection.

3. The relationship to tuberculosis, pyogenic infections and steroid therapy and rheumatoid disease is discussed.

William B. Seaman
622 West 168th Street
New York 32, New York

We wish to acknowledge the cooperation of the staff of the Arthritis Clinic in the preparation of this paper.

REFERENCES

1. ANDERSSON, O. Cited by Romanus and Ydén.[20]
2. ANSELL, G. Complications of adreno-cortical therapy; radiological survey. *J. Fac. Radiologists*, 1958, *9*, 113–126.
3. BAGGENSTOSS, A. H., BICKEL, W. H., and WARD, L. E. Rheumatoid granulomatous nodules as destructive lesions of vertebrae. *J. Bone & Joint Surg.*, 1952, *34-A*, 601–609.
4. BLUMBERG, B., and RAGAN, C. Natural history of rheumatoid spondylitis. *Medicine*, 1956, *35*, 1–31.
5. COMPERE, E. L., and GARRISON, M. Correlation of pathologic and roentgenologic findings in tuberculous and pyogenic infections of vertebrae; fate of intervertebral disk. *Ann. Surg.*, 1936, *104*, 1038–1067.
6. COUTTS, M. B. Atlanto-epistropheal subluxations. *Arch. Surg.*, 1934, *29*, 297–311.
7. EDSTRÖM, G. Is spondylarthritis ankylopoietica independent disease or rheumatic syndrome? *Acta med. scandinav.*, 1940, *104*, 396–413.
8. FORESTIER, J., JACQUELINE, F., and ROTES-QUEROL, J. Ankylosing Spondylitis: Clinical Considerations, Roentgenology, Pathologic Anatomy, Treatment. Translation by Desjardins, A. U. Charles C Thomas, Publisher, Springfield, Ill., 1956, p. 249.
9. GARCIA, A., JR., and GRANTHAM, S. A. Hematogenous pyogenic vertebral osteomyelitis. *J. Bone & Joint Surg.*, 1960, *42-A*, 429–436.
10. GUEST, C. M., and JACOBSON, H. G. Pelvic and extrapelvic osteopathy in rheumatoid spondylitis; clinical and roentgenographic study of ninety cases. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 760–768.
11. HEIMANN, W. G., and FREIBERGER, R. H. Avascular necrosis of femoral and humeral heads after high-dosage corticosteroid therapy. *New England J. Med.*, 1960, *263*, 672–675.
12. LAME, E. L. Vertebral osteomyelitis following operation on urinary tract or sigmoid; third lesion of uncommon syndrome. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *75*, 938–952.
13. MARGULIES, M. E., KATZ, I., and ROSENBERG, M. Spontaneous dislocation of atlanto-axial joint in rheumatoid spondylitis. *Neurology*, 1955, *5*, 290–294.
14. MARTEL, W. Occipito-atlanto-axial joints in rheumatoid arthritis and ankylosing spondylitis. To be published.
15. MURRAY, R. O. Radiological bone changes in Cushing's syndrome and steroid therapy. *Brit. J. Radiol.*, 1960, *33*, 1–19.
16. PRATT, T. L. Spontaneous dislocation of atlanto-axial articulation occurring in ankylosing spondylitis and rheumatoid arthritis. *J. Fac. Radiologists*, 1959, *10*, 40–43.
17. PURSER, D. W., and SHARP, J. Spontaneous atlanto-axial dislocation, complication of spondylitis. *J. Bone & Joint Surg.*, 1957, *39-B*, 582.
18. RAGAN, C. Personal communication.

19. Rand, C. W. The Neurological Patient: His Problems of Diagnosis and Care. Charles C Thomas, Publisher, Springfield, Ill., 1944, p. 47.
20. Romanus, R., and Ydén, S. Pelvo-Spondylitis Ossificans: Rheumatoid or Ankylosing Spondylitis. Year Book Publishers, Chicago, 1955.
21. Sharp, J., Purser, D. W., and Lawrence, J. S. Rheumatoid arthritis of cervical spine in adult. *Ann. Rheum. Dis.*, 1958, *17*, 303.
22. Steiner, R., Cited by Murray.[15]
23. Watson-Jones, R. Fractures and Joint Injuries. Fourth edition. Williams & Wilkins, Baltimore, 1952, p. 981.
24. Werne, S. Spontaneous dislocation of atlas as complication in rheumatoid arthritis. *Acta rheum. scandinav.*, 1957, *3*, 101–107.
25. Wholey, M. H., Pugh, D. G., and Bickel, W. H. Localized destructive lesions in rheumatoid spondylitis. *Radiology*, 1960, *74*, 54–56.

VOL. 86, No. 2

OSTEOLYSIS OF THE ACROMIAL END OF THE CLAVICLES IN RHEUMATOID ARTHRITIS*

By MEYER ALPERT, M.D., *and* MORTON MEYERS, M.D.
NEW YORK, NEW YORK

DESTRUCTIVE changes in the outer end of the clavicles have been observed in 11 patients with advanced rheumatoid arthritis. Since these osseous manifestations have not been adequately described previously, it is the purpose of this paper to present these findings.

REVIEW OF THE LITERATURE

A survey of the medical literature of the past seventy-five years brought to light very little information on the clavicular changes in rheumatoid arthritis. In over 150 books and articles dealing with rheumatoid arthritis and disorders of the shoulder, we found only 2 reports[15,22] which mentioned significant changes in the outer ends of the clavicles.

In 1949 Layani *et al.*[22] published the case of a sixty-five year old woman who had had rheumatoid arthritis involving many peripheral articulations for a seventeen year period. Joint motion was so painful that she was confined to bed or a chair. On physical examination the elbows were fixed, and there was shortening of the phalanges. Roentgen examination showed severe demineralization of the entire skeleton, with advanced changes due to rheumatoid arthritis in the hands, wrists, knees and hips. Tapering of the outer ends of the clavicles and widening of the acromioclavicular joint spaces were noted. In 1953 Jacobs *et al.*[15] described the case of a sixty-three year old woman who had suffered from widespread rheumatoid arthritis for twenty-eight years. Roentgen examination showed striking arthritic changes in the hands, wrist, elbows, knees, ankles and feet. Jacobs and his co-workers stated that there was a loss of bone substance of the outer end of the left clavicle. The illustrations in this article show that this patient also had a similar involvement of the outer end of the right clavicle.

Kienböck's comprehensive "Röntgendiagnostik der Knochen und Gelenkkrankheiten" does contain a description of 3 patients (No. 120, 122 and 123) who had destructive changes in the outer ends of the clavicles, which were, in all likelihood, secondary to rheumatoid arthritis. Unfortunately, Kienböck diagnosed the first case as rheumatoid tuberculosis, the second as rheumatoid tuberculosis (Poncet) and the third as rheumatoid tuberculosis with caries sicca. It should be mentioned that in Kienböck's era the Austrian pathologists seriously considered tuberculosis as the etiology of the affection we now call rheumatoid arthritis.[1,3,31]

That acromioclavicular joint changes may take place in rheumatoid arthritis was known by Oppenheimer,[30] who in 1944 stated that roentgenograms may demonstrate narrowing of the joint space and erosion of the articular surfaces. He found that the arthritic process may progress to bony ankylosis, but in other patients the destructive process stops, and sclerotic areas appear in the articular areas, thereby simulating osteoarthritis. In 1954 Laine *et al.*[21] studied the records of 277 hospitalized patients with rheumatoid arthritis and found that 57.4 per cent had shoulder symptoms. Only one of these patients, however, had acromioclavicular arthritis, and the roentgen findings were not described. In Schinz's "Roentgen-Diagnostics" "erosion of the articular surfaces" is mentioned in the text. The authors of two books dealing exclusively with arthritis of the shoulder[24,41] do not discuss the alterations of the clavicle. Three other recent

* From the Radiological Service of the Presbyterian Hospital, New York, New York, and the Department of Radiology, College of Physicians and Surgeons, Columbia University.

TABLE I

PERTINENT FINDINGS IN 11 PATIENTS WITH CLAVICULAR OSTEOLYSIS DUE TO RHEUMATOID ARTHRITIS

Case No.	Age*	Sex	Duration Rheumatoid Arthritis (yr.)	Shoulder Symptoms and Signs	Other Joint Symptoms	Physical Findings in Other Joints	Roentgen Findings in Other Joints	Laboratory Data
I	47	M	18	None	Progressive deformity and disability of peripheral joints	Marked flexion deformities and contractions of all peripheral joints. Fingers in ulnar deviation. Wrists fused. Decreased mobility of temporomandibular joints	Knees, elbows, hands and wrists, feet and ankles: demineralization flexion contractures with ulnar deviation, destruction of articular cortices, subluxation of metacarpophalangeal and knee joints. Otto pelvis, fusion of sacroiliac and manubriosternal joints. Marked deformity of femoral heads	E.S.R. 80 Latex fixation test positive
II	65	F	45	None	Moderate pain and progressive deformity in hands, feet, ankles, knees, wrists, elbows and hips	All distal joints deformed, with contractures at elbows. Right wrist fused. Severe ulnar deviation of fingers. Hip flexion only to 20°	None available	E.S.R. 32. Latext fixation test positive
III	67	M	6	Limitation of motion and deformity of shoulders	Progressive deformity of small joints, and limitation of motion of all large joints, including knees and elbows	Decreased motion in eblows, knees and ankles. Knees swollen and warm. Ulnar deviation of fingers. Subcutaneous nodules over elbows and wrists. Spine moderately stiff. General muscle atrophy	Hands and knees: demineralization, gross erosion and destruction of articular surfaces, numerous cyst-like rarefactions, narrowed joint spaces. Subluxation of metacarpophalangeal joints	E.S.R. 38
IV	31	F	9	Progressive ankylosis of shoulders	Progressive stiffness and flexion contractures of knees, ankles, elbows, hips, and jaws	Extremities emaciated, with ulnar deviation of fingers. Flexion contractures and markedly restricted motion of joints	Right wrist and knee fused. Severe periarticular demineralization, marked erosion and destruction of all articular surfaces; cyst-like areas of rarefaction, and narrowed joint spaces in knees, ankles, elbows and hips	None available
V	47	F	24	None	Progressive deformity and disability of all peripheral joints	Marked flexion deformities and contractures of hands, ankles, feet and knees, with limitation of motion mainly in hands. Ulnar deviation of fingers. Fusion of wrists	Ankylosis of carpal bones. Advanced demineralization, articular destruction, and subluxation of phalanges of hands and feet; joint space narrowing in hands, feet and knees. Pseudocyst in head of fifth metatarsal	E.S.R. 41
VI	58	F	26	Limited motion of shoulders	Progressive flexion contractures of left hip, wrists and knees	Contractures, limitation of motion and crepitation in left hip, knees and wrists. Subcutaneous nodules over elbows	None available	None available
VII	45	F	23	Frozen right shoulder	Progressive painful stiffness and deformities of many joints, most marked in hands	Joint deformities in hand with ulnar deviation of motion, and small subcutaneous nodules. Elbows stiff. Fluid in knee joints	Elbows, wrists, hands, knees, ankles, feet: flexion contractures with ulnar deviation, demineralization, destruction of articular cortices, subluxation of metacarpophalangeal joints	E.S.R. 42. Latext fixation test positive. Sheep cell agglutination positive

Case No.	Age*	Sex	Duration of Rheumatoid Arthritis (yr.)	Shoulder Symptoms and Signs	Other Joint Symptoms	Physical Findings in Other Joints	Roentgen Findings in Other Joints	Laboratory Data
VIII	61	M	18	None	Progressive deformity and disability of hands, wrists, feet and ankles	Marked flexion contractures and deformities of hands and elbows. Subcutaneous nodules.	Knees: demineralization, narrow joint spaces, destruction of articular cortices, subluxation	E.S.R. 41. Latex fixation test positive
IX	49	M	14	Limitation of motion of left shoulder and atrophy of left shoulder girdle	Chronic progressive deformity and disability of right fingers, ankles, and knees	Wrists, knees, ankles, first and second right fingers: ulnar deviation, fusiform swelling, flexion contractures	Hands, wrists, knees, ankles, and feet: loss of articular cortices, joint space narrowing, demineralization, cyst-like areas of rarefaction, periarticular swelling	E.S.R. 150. Sheep cell agglutination positive
X	35	F	22	None	Progressive deformity and disability of peripheral joints	Flexion contractures of elbows, wrists and fingers. Fusiform deformity and ulnar deviation of fingers	Knees, feet, elbows: flexion contractures, narrow joint spaces, demineralization pseudocyst formation, destruction of articular cortices	E.S.R. 22
XI	56	M	10	None	Progressive deformity and disability of hands, wrists, elbows, knees and feet	Hands, wrists, elbows, knees, feet: flexion contractures, periarticular Ulnar deviation of fingers	Hands and wrists: narrow joint spaces, demineralization, loss of articular cortices, cyst-like areas of rarefaction	E.S.R. 116. Latex fixation test positive

* Age of patient at the time of last visit.

publications[14,43,55] contain roentgenograms demonstrating the clavicular tapering, but without comments from their authors. Erosions of the acromial ends of the clavicles have been described in 2 patients with severe psoriatic arthritis.[17,42] Unilateral acromioclavicular arthritis has also been observed in one patient with long-standing Marie-Strümpell arthritis.[5] Involvement of the sternoclavicular joints by rheumatoid arthritis has been studied by several authors,[18,35,44,48] and this affection will not be discussed in the present paper.

CLINICAL MATERIAL

In our study of 11 patients with clavicular changes, the average age was fifty-one years, with a range of thirty-one to sixty-seven. There were 6 women and 5 men. The average duration of the arthritic symptoms was 19.5 years, with a minimum of six and a maximum of forty-five years (Table 1). Five of the 11 patients had complaints referable to the shoulder, but the medical records did not show implication of the acromioclavicular joints. The usual symptoms were pain and stiffness in multiple peripheral articulations, and the common physical findings included ulnar deviation of the fingers, flexion deformities, decreased motion and swelling of the joints. In 9 patients the erythrocyte sedimentation test had been done, and in each the rate was elevated, with a variation from 22 to 150. In 6 patients a laboratory test for rheumatoid arthritis[32,38,50] had been carried out (sheep cell agglutination or latex fixation), and in 5 it was positive. The patient with the negative latex fixation test (Case x) had characteristic clinical and roentgen manifestations of long-standing peripheral rheumatoid arthritis, the disease having started at thirteen years of age. This test had been done only once, when she was thirty-five years old, and at that time she was clinically in a state of remission. Her sedimentation rate (Westergren) was only 22 mm. per hour, and this lack of activity probably explains the low titer.[38]

TABLE II

ROENTGEN FINDINGS IN OUTER END OF CLAVICLE IN 11 PATIENTS WITH CLAVICULAR OSTEOLYSIS DUE TO RHEUMATOID ARTHRITIS

Case No.	Right Clavicle	Left Clavicle	Acromioclavicular Angle	
			Right	Left
I	Marked loss of bone substance inferiorly, with widening of the joint space (15 mm.). Demineralization	Demineralization, with narrowing of the joint space and an osteophytic spur	28°	42°
II	Slight demineralization. Irregular margins. Decreased joint space	Loss of bone substance inferiorly, with small area of sclerosis	30°	10°
III	Truncated with irregular margins	Loss of bone substance inferiorly, with widening of joint space	25°	31°
IV	Distal tapering with loss of bone substance inferiorly		44°	27°
V	Loss of bone substance inferiorly, producing tapering with widening of the joint space (15 mm.). Progressive changes over 12 year course		33°	45°
VI	Loss of bone substance inferiorly, producing tapering, and widened joint space. Progressive changes over 9 year course		37°	66°
VII	Loss of bone substance producing tapering		52°	29°
VIII	Bulbous appearance with scalloping, progressing to "ace-of-spades" appearance over 2 year course	Progressive scalloping	40°	32°
IX	No changes	Loss of bone substance inferiorly	0°	45°
X	Notching	No changes	32°	14°
XI	Scalloping	Loss of bone substance inferiorly producing tapering, with demineralization	35°	28°

ROENTGEN FINDINGS

The roentgenograms in 7 of our patients revealed a striking loss of bone substance at the acromial end of each clavicle. The other 4 cases showed only cortical erosion and pseudocyst formation (Table II). In severe cases the outer end of the clavicle often had a tapered appearance, but bone destruction was more marked inferiorly than superiorly. Accompanying changes of the acromion process, characterized by demineralization and articular erosion, were less marked and more difficult to recognize. The acromioclavicular joint space was definitely widened in 6 patients. Two patients had unilateral narrowing of this joint space due to secondary degenerative alterations. The osseous structures of the shoulder girdle showed severe osteoporosis. Involvement of the glenohumeral joint by rheumatoid arthritis was seen in 9



FIG. 1. Case V. (1947) Roentgenogram of a thirty-five year old woman who has had rheumatoid arthritis for twelve years showing a normal outer end of the right clavicle.

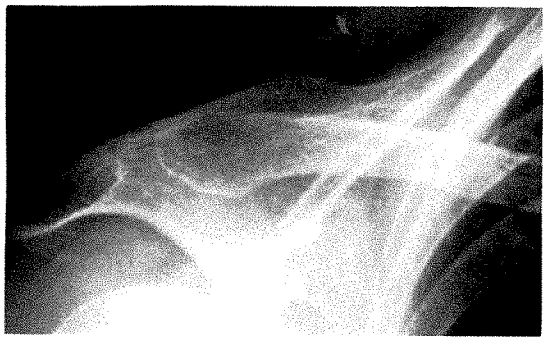


FIG. 2. Case V. (1955) Roentgenogram showing loss of bone substance of the outer end of the right clavicle, especially inferiorly.

of the 11 patients, and many of the peripheral joints showed evidence of long-standing rheumatoid arthritis.

In one patient (Case V, Fig. 1, 2 and 3) we were able to follow the unfavorable progress of the clavicular lesion from a normal status in 1947 to a definite loss of bone substance of the acromial end in 1953. By 1955 the tapering had increased, and in 1959 there was a 15 mm. gap at the acromioclavicular joints due to continued clavicular bone loss. Another patient (Case VI, Fig. 4) had progressive tapering of the outer ends of the clavicles over a nine year interval; and still another (Case VII, Fig. 5) showed a beaked appearance of the outer end of the right clavicle with a fifteen year history of rheumatoid arthritis.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of a destructive lesion of the outer end of the clavicle,



FIG. 3. Case V. (1959) Roentgenogram showing further tapering of the outer end of the right clavicle and marked widening of the acromioclavicular joint space.



FIG. 4. Case VI. Roentgenogram of a fifty-eight year old woman who has had rheumatoid arthritis for twenty-six years showing considerable tapering of the outer end of the right clavicle.

there are many conditions to consider: normal variation of the acromial end of the clavicle, cleidocranial dysostosis, osteogenesis imperfecta, hyperparathyroidism, acromioclavicular dislocation, post-traumatic osteolysis of the clavicle, surgical resection of the outer end of the clavicle, inflammatory diseases, degenerative changes, scleroderma, acroosteolysis and neoplastic lesions.

Normal Clavicle. The normal clavicle was examined in over 300 adult patients to evaluate the roentgen appearance of the acromial end. This study confirmed the fact that there is considerable variation in the configuration of the outer end of this bone. Although many individuals had expansion of this segment of the clavicle, we found only one patient who had any appreciable tapering of this region. On the anteroposterior roentgenogram of the shoulder, the plane of the normal acromio-



FIG. 5. Case VII. Roentgenogram of a thirty-seven year old woman who has had rheumatoid arthritis for fifteen years showing a beaked appearance of the outer end of the right clavicle due to bone loss both superiorly and inferiorly.

clavicular joint is usually angulated with respect to the sagittal plane, so that the inferior aspect of the articulation is more medially situated than the superior part. It is especially this type which must be differentiated from erosion of the inferior aspect due to rheumatoid arthritis.

DePalma[4] used the magnitude of this angle as a criterion to classify all clavicles into 3 categories. Type 1 had an average angle of 16°, Type 2—24.6° and Type 3—36.1°. Of the 66 clavicles studied by De Palma 89 per cent were either Type 1 or Type 2, and only 11 per cent were Type 3. In our 11 patients with rheumatoid arthritis, there were only six clavicles that fell into Types 1 and 2, while 8 were of Type 3 variety. These figures indicate a reversal of the normal distribution. Seven clavicles had such high angles (ranging from 40° to 66°) that they could not be placed in any of DePalma's categories. One clavicle had an angle of 0°, indicating that it was parallel to the sagittal plane. The average angle of the 22 clavicles in our study was 33° (Table II).

The normal acromioclavicular joint space is 2–5 mm. in diameter,[30] but in rheumatoid arthritic involvement this gap may be increased to 15 mm. (Case 1).

Osteogenesis Imperfecta. In osteogenesis imperfecta one may see a slender clavicle bilaterally, with tapering of the outer ends. In contrast to stiff joints, however, these patients often have hypermobile articulations. The presence of fresh fractures, deformities due to old fractures, and bowing of the long bones will aid in the differential diagnosis. Blue sclerae are usually present, and there is sometimes a family history of this disorder.

Hyperparathyroidism. Erosive changes in the outer ends of the clavicles are important findings in the roentgen diagnosis of hyperparathyroidism.[9,28,47] In both hyperparathyroidism and rheumatoid arthritis, the loss of bone substance is characteristically bilateral, and there is demineralization of the regional osseous structures. As a rule neither condition produces clavicular pain, but there may be a local tumefaction at the site of the lesion in hyperparathyroidism. In hyperparathyroidism we have not observed sharp tapering of the clavicle. In patients with hyperparathyroid bone disease, there will be subperiosteal cortical resorption of the phalanges,[8,34] and most of them will have definite skull changes,[7] including demineralization of the skull tables and loss of the lamina dura. The presence of renal calculi or nephrocalcinosis will favor the diagnosis of hyperparathyroidism.

Acromioclavicular Dislocation. The usual case of an acute acromioclavicular dislocation[46] will not constitute a difficult problem in differential diagnosis, as there will be a history of trauma, and the patient will complain of pain in the joint region. On both physical and roentgen examination, the outer end of the clavicle will be displaced superiorly, a finding not present in rheumatoid arthritis. We have observed several cases of chronic acromioclavicular dislocations in which there was a considerable gap in the acromioclavicular joint space due to rupture of the acromioclavicular ligaments, and this finding may resemble that seen in rheumatoid arthritis. Factors to be considered, however, in differential diagnosis include the past history of a severe injury, the unilaterality of the lesion, the presence of bone sclerosis or deformity secondary to an old healed fracture. Often there is also evidence of calcification or ossification of the torn coracoacromial and acromioclavicular ligaments.

Post-Traumatic Osteolysis. Post-traumatic osteolysis[16,54] involves the clavicle more often than any other bone, and the outer end is the site of predilection. It is readily apparent that the dissolution of the acromial end of the clavicle will simulate the changes seen in rheumatoid arthritis. Post-traumatic osteolysis, however, is a unilateral affection, usually occurring in young adult males. The bone destruction may slowly progress to involve the adjacent scapula and ribs, while costal extension does not occur in rheumatoid arthritis. A biopsy may be necessary for diagnosis, and the histologic appearance is fre-

quently that of hemangiomatosis.[10] It might also be mentioned in this connection that unilateral erosion of the outer end of the clavicle has been described as an occupational disease in men using compressed air tools,[6] evidentally on the basis of repeated minor injuries.

Surgical Resection. A resection of the outer end of the clavicle[51] may result in a roentgen appearance resembling the clavicular deformity of rheumatoid arthritis. In one of our patients (Case XI) with rheumatoid arthritis, the clavicular defect was erroneously interpreted as being a postoperative sequel. A sharply tapering clavicle is not apt to be of surgical origin.

Tuberculosis. Tuberculosis may produce erosion of the outer end of the clavicle, thereby simulating the lesion seen in rheumatoid arthritis. In some cases of clavicular tuberculosis, moreover, the acromion is not appreciably involved by the destructive process, and there may be no soft tissue swelling and very little discomfort.[39,46] Unilateral involvement would favor the diagnosis of tuberculosis, while polyarthritis would favor rheumatoid arthritis. If the acromion is severly involved, if there is a painful soft tissue mass and if there is a draining sinus, tuberculosis is undoubtedly present. Glenohumeral joint involvement is not apt to be associated with a clavicular lesion in tuberculosis, but this association is common is rheumatoid arthritis. Aspiration or biopsy of the lesion may be required, if tuberculosis is suspected.

Other Inflammatory Lesions. Inflammatory lesions other than tuberculosis rarely affect the clavicle; however, this has been reported in cases of pyogenic osteomyelitis, Brodie's abscess, mycotic infection, gumma and eosinophilic granuloma involving the bone.[2,11,36] All five lesions are unilateral and will often be accompanied by soft tissue swelling, but the latter is absent in rheumatoid arthritis. Subperiosteal new bone formation is present in a pyogenic osteomyelitis and may also be seen in eosinophilic granuloma, but this change will not be evident in rheumatoid arthritis. In the case of gumma, the Wassermann test will be of considerable diagnostic value.

Degenerative Arthritis of the Acromioclavicular Joints. Degenerative arthritis of the acromioclavicular joint is encountered twice as often as all other disorders of this articulation combined.[13,26,27] The roentgen appearance is considerably different from that of rheumatoid arthritis, for there is sclerosis, osteophyte formation and narrowing of the joint space.

Scleroderma. Meszaros mentioned in his recent review on scleroderma that one patient had a loss of bone substance at the acromial end of each clavicle. This osseous abnormality is very rare in scleroderma, and we were unable to find a similar case reported in the literature. Despite the presence of the clavicular lesion, there should not be any difficulty in distinguishing scleroderma from rheumatoid arthritis. The presence of Raynaud's phenomenon, hardened facial skin with immobile expression, ulceration of the finger-tips and dysphagia are clinical features which would favor the diagnosis of scleroderma. The following roentgen features are also useful in supporting this impression: loss of the manual ungual tufts, calcinosis and atony of the esophagus.

Acroosteolysis. There are several reports in the literature indicating that patients with acroosteolysis may have erosions of the outer end of both clavicles.[12,20,52] In these cases the shortening of the fingers may simulate the "opera glass hand" of mutilating rheumatoid arthritis; however, these patients do not have a history of polyarthralgias associated with soft tissue swelling and warmth of the affected articulations. Nor do they have pain or limitation of motion in the larger peripheral joints. The roentgen examination will show a progressive loss of bone substance of the ungual tufts, then of the entire distal phalanges and finally of the middle phalanges, as well. Both hands and both feet are characteristically involved, but the joints are not the first structures to be affected.

Neoplasms. Both primary and secondary neoplasm of the clavicles are rare,[33] and

one would not expect the outer end of both clavicles to be involved in a single patient. The presence of a painful mass will be against the diagnosis of rheumatoid arthritis, and if there is a question of tumor, a biopsy is often indicated.

DISCUSSION

The pathology of the clavicular changes in rheumatoid arthritis is not known, as we do not have any biopsies of this region. However, since at autopsy the outer end of the clavicle is available for examination, we hope that these changes will be clarified in the near future. In all probability the erosion of the clavicle is similar to the loss of bone seen in mutilating arthritis. Here, gross and microscopic examination has demonstrated replacement of the involved bone end, articular cartilage, synovial membrane and a considerable portion of the muscle by fat[23] or fibrous tissue.[53] These pathologic findings evidently represent the residue of a chronic inflammatory process due to rheumatoid arthritis.

SUMMARY

The erosive changes in the outer ends of the clavicles in cases of rheumatoid arthritis have been described. The clavicle is tapered or scalloped due to a loss of bone substance, and both clavicles are often involved. The acromioclavicular joint space may be widened, but the acromion process is not severely affected. This clavicular lesion must be distinguished from bone destruction caused by hyperparathyroidism, tuberculosis, post-traumatic osteolysis and acroosteolysis.

Meyer Alpert, M.D.
Department of Radiology
Presbyterian Hospital
622 West 168th Street
New York 32, New York

REFERENCES

1. BRAV, E. A., and HENCH, P. S. Tuberculous rheumatism; a résumé. *J. Bone & Joint Surg.*, 1934, *16*, 839–866.
2. CONWAY, F. M. Syphilis of clavicle. *Ann. Surg.*, 1934, *99*, 290–300.
3. COPEMAN, W. S. C., and CLAY, R. D. Rheumatoid arthritis believed to be of tuberculous origin; report of 2 cases. *Lancet*, 1935, *2*, 1460–1461.
4. DEPALMA, A. F. Degenerative Changes in the Sternoclavicular Joint and Acromioclavicular Joints in Various Decades. Charles C Thomas, Publisher, Springfield, Ill., 1957.
5. ENGFELDT, B., ROMANUS, R., and YDÉN, S. Histologic studies of pelvo-spondylitis ossificans (ankylosing spondylitis) correlated with clinical and radiologic findings. *Ann. Rheum. Dis.*, 1954, *13*, 219–228.
6. EHRICHT, H. G. Die Osteolyse im lateralen Claviculaende nach Pressluftschaden. *Arch. Orthop. Chir.*, 1959, *50*, 576–582.
7. ELLIS, K., and HOCHSTIM, R. J. Skull in hyperparathyroid bone disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 732–742.
8. FRIEDENBERG, R. M., and SAYEGH, V. Advanced skeletal changes in hyperparathyroidism. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 743–747.
9. FRIEDMAN, R. L., RUSSI, D., and BARRY, W. F., JR. Clavicular changes associated with secondary hyperparathyroidism. *Virginia M. Month*, 1954, *81*, 761–767.
10. GORHAM, L. W., and STOUT, A. P. Massive osteolysis (acute spontaneous absorption of bone, phantom bone, disappearing bone); its relation to hemangiomatosis. *J. Bone & Joint Surg.*, 1955, *37-A*, 985–1004.
11. GRAY, F. Osteomyelitis of clavicle. *Brit. J. Surg.*, 1945, *32*, 466–467.
12. HARNASCH, H. Die Akroosteolysis, ein neuer Krankheitsbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1950, *72*, 352–359.
13. HECK, C. J., and CHANDLER, F. A. Diagnosis and treatment of pain in acromioclavicular joint. *J. Am. Geriat. Soc.*, 1955, *3*, 993–996.
14. HOLLANDER, J. L. Arthritis and Allied Conditions. Sixth edition. Lea & Febiger, Philadelphia, 1960, p. 125.
15. JACOBS, E., BASTENIE, P. A., and MEYERS, A. Arthrite mutilante avec ostéolyse progressive des membres supérieurs. *Acta clin. belg.*, 1953, *8*, 444–450.
16. JONES, G. B., MIDGLEY, R. L., and SMITH, G. S. Massive osteolysis-disappearing bones. *J. Bone & Joint Surg.*, 1958, *40-B*, 494–501.
17. JUNGMANN, H., and STERN, V. S. Unusual case of joint disease (possible example of arthritis psoriatica). *Brit. J. Radiol.*, 1944, *17*, 383–385.
18. KHALAF, S. Sur les arthropathies sterno-claviculaires. *Rev. Méd. Moyen Orient*, 1958, *15*, 414–423.
19. KÖHLER, A., and ZIMMER, E. A. Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology. English translation by Case, J. T. Grune & Stratton, New York, 1956, pp. 171–177.

20. LACROIX and BOTELLA, A. A propos d'un cas d'epiphysiolyse essentielle. *J. de radiol. et d'électrol.*, 1954, *35*, 626–628.
21. LAINE, V. A. I., VAINIO, J. J., and PEKANMÄKI, K. Shoulder affections in rheumatoid arthritis. *Ann. Rheum. Dis.*, 1954, *13*, 157–160.
22. LAYANI, F., DJIAN, A., and FELSENWALD, C. Ostéoporose diffuse et intense avec ostéolyse bilaterale et symetrique des segments proximaux et distaux. *Rev. rhum.*, 1949, *16*, 156–160.
23. MARIE, P., and LERI, A. Une variété rare de rhumatisme chronique: la main en lorgnette. *Bull. et mém. Soc. méd. hôp. Paris*, 1913, *36*, 104–107.
24. MERKLEN, R. Les Rhumatismes de l'Épaule. G. Doin, Paris, 1937.
25. MESZAROS, W. T. Regional manifestations of scleroderma. *Radiology*, 1958, *70*, 313–325.
26. MICHOTTE, L. J. L'arthrose acromio-claviculaire. *Acta orthop. belg.*, 1952, *18*, 181–183.
27. NATHAN, W. Chronische, deformierende Erkrankungen des Akromioklavikulargelenks. *München. med. Wchnschr.*, 1932, *79*, 2007–2009.
28. NATHANSON, L., and SLOBODKIN, M. Acromioclavicular changes in primary and secondary hyperparathyroidism. *Radiology*, 1950, *55*, 30–35.
29. OPPENHEIMER, A. Arthritis of acromioclavicular joint. *J. Bone & Joint Surg.*, 1943, *25*, 867–870.
30. OPPENHEIMER, A. Lesions of acromioclavicular joint causing pain and disability of shoulder. AM. J. ROENTGENOL. & RAD. THERAPY, 1944, *51*, 699–706.
31. PONCET, A. Rhumatisme tuberculeux ankylosant. *Bull. et mém. Soc. méd. hôp. Paris*, 1903, *20*, 841–849.
32. PLOTZ, C. M., and SINGER, J. M. Latex fixation test. II. Results in rheumatoid arthritis. *Am. J. Med.*, 1956, *21*, 893–896.
33. PRATT, G. F., DAHLIN, D. C., and GHORMLEY, R. K. Tumors of scapula and clavicle. *Surg., Gynec. & Obst.*, 1958, *106*, 536–544.
34. PUGH, D. G. Subperiosteal resorption of bone; roentgenologic manifestation of primary hyperparathyroidism and renal osteodystrophy. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *66*, 577–586.
35. RATHCKE, L. Beitrag zur normalen und pathologischen Anatomie des Sternoclaviculargelenkes. *Deutsche Ztschr. Chir.*, 1937, *249*, 162–169.
36. RAY, R. B., and KELLNER, A. Eosinophilic granuloma of bone; report of case involving clavicle. *J. Bone & Joint Surg.*, 1946, *28*, 629–634.
37. RONOT, P. Contribution à l'étude des formes ostéolytiques de la polyarthrite chronique rhumatismale. Thèse 181 de la Faculté Mixte de Médecine et de Pharmacie de Lyon, 1957.
38. ROSE, H. M., RAGAN, C., PEARCE, E., and LIPMAN, M. O. Differential agglutination of normal and sensitized sheep erythrocytes by sera patients with rheumatoid arthritis. *Proc. Soc. Exper. Biol. & Med.*, 1948, *68*, 1–6.
39. ROSENDAHL-JENSEN, S. Tuberculous osteomyelitis of clavicle—report of five cases. *Acta. chir. scandinav.*, 1960, *118*, 327–330.
40. SCHINZ, H. R., BAENSCH, W. E., FRIEDL, E., and UEHLINGER, E. Roentgen Diagnostics. English translation by Case, J. T. Grune & Stratton, New York, 1952, p. 1989.
41. DE SÈZE, S., RYCKEWAERT, A., and MAITRE, M. L'Épaule en Pratique Rhumatologique. Masson & Cie, Paris, 1959, pp. 74–91.
42. SHLIONSKY, H., and BLAKE, F. G. Arthritis psoriatica: report of a case. *Ann. Int. Med.*, 1936, *10*, 537–546.
43. SMYTH, C. J. Bone absorption in rheumatoid arthritis. In: Rheumatic Diseases. American Rheumatism Association. W. B. Saunders Company, Philadelphia, 1952, pp. 196–208.
44. SOKOLOFF, L., and GLEASON, I. O. The sternoclavicular articulation in rheumatic diseases. *Am. J. Clin. Path.*, 1954, *24*, 406–414.
45. STOCKS, P., and BARRINGTON, A. Heriditary disorders of bone development. In: Treasury of Human Inheritance. Edited by Karl Pearson. Vol. III. Cambridge University Press, London, 1925, p. 123.
46. TAUB, S. J., FRIEDMAN, I. A., and DIBBLE, J. B. Tuberculosis of acromioclavicular joint with subscapular bursitis and effusion. *Illinois M.J.*, 1950, *98*, 357–360.
47. TENG, C. T., and NATHAN, M. H. Primary hyperparathyroidism. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 716–731.
48. TREANOR, W., GRANT, A., and PSOKI, R. Unusual sites of rheumatoid arthritis. *Arch. Phys. Med.*, 1956, *37*, 699–705.
49. URIST, M. R. Complete dislocations of acromioclavicular joint. *J. Bone & Joint Surg.*, 1946, *28*, 813–837.
50. WAALER, E. On occurrence of factor in human serum activating specific agglutination of sheep blood corpuscles. *Acta path. et microbiol. scandinav.*, 1940, *17*, 172–188.
51. WAGNER, C. J. Partial claviculectomy. *Am. J. Surg.*, 1953, *85*, 259–265.
52. WASSNER, U. J. Ein weiterer Fall einer Akroosteolyse —zugleich ein Beitrag zu ihrer Differentialdiagnose. *Fortsch. a. d. Geb. d. Röntgenstrahlen*, 1954, *80*, 186–191.
53. WERTHEMANN, A. Pied en lorgnette, arthritis mutilans. *Schweiz. med. Wchnschr.*, 1945, *75*, 749–751.
54. WILNER, S. Disappearing bones. *J. Bone & Joint Surg.*, 1958, *40-B*, 502–513.
55. ZADEK, I., and BUDNICK, M. Polyarthritis mutilans. *N. York State J. M.*, 1959, *59*, 1125–1130.

THE OCCIPITAL VERTEBRA

By G. LOMBARDI, M.D.*
MILANO, ITALY

ACCORDING to the investigations of Froriep, the basilar process of the occipital bone is formed by fusion of the first three primitive vertebrae (or occipitoblasts), the most caudal of which is the so-called occipital vertebra (Froriep) or proatlas (Albrecht). A theory advanced by Chiarugi in 1895 and later developed by Kollmann in 1905 attributes failure of the distal occipitoblast to fuse with the others as the cause of abnormal bone formations on the external surface of the skull around the occipital foramen. This phenomenon is called the "manifestation of the occipital vertebra."

This malformation, described for the first time at the beginning of the 19th century, has been the subject of numerous studies in normal and comparative anatomy and anthropology. The great interest derives from the fact that the occipitovertebral joint, just as the lumbosacral joint, is in an embryologically unstable region. In the vertebrates, the border between the cranium and the vertebral column moves in the caudal direction and reaches its lowest position in the mammals. In man the instability of this border is observed in cases of assimilation of the atlas and is to be regarded as a progressive variant, while in cases of manifestation of the occipital vertebra it is to be regarded as a recessive trait.

The concept of the manifestation of the occipital vertebra and its limits have not as yet been clearly defined. Schmidt and Fischer[16,17] remark that there is hardly a variant of the occipital bone or of the cranial part of the atlas which has not been attributed to it. We have included: (a) the third condyle; (b) the paracondyloid process; (c) the basilar processes; (d) the accessory bone elements, separate or fused to the foramen magnum; and (e) the transverse fissures of the basioccipital bone.

From the anatomic point of view, this classification is not complete; some variants have been omitted either because they are not demonstrable roentgenologically (such as the division of the foramen of the hypoglossal nerve) or because they can be classified among the anatomic variants (such as the ponticulus posterius which, closing the sulcus of the vertebral artery of the atlas, forms the arched foramen).

We have found only a few articles concerning this malformation in the roentgenologic literature. The first one, published by Hadley[7] in 1948, has been followed by a few others, which deal mostly with isolated observations. Exceptions to this are the publication of McRae and Barnum[14] on 5 paracondyloid processes observed among 25 cases of assimilation of the atlas, the study of Schmidt,[16] carried out partly in the living subject and partly on anatomic material, the report of Fischer[5] and the recent work by Schmidt and Fischer[17] on occipital dysplasia.

The present paper concerns the manifestation of the occipital vertebra based on roentgen examinations of 4,000 consecutive patients. The collected cases of this malformation were 19 in number—a high incidence if we consider the limitations of the roentgen examination, however accurately it is performed.

ROENTGEN TECHNIQUE

The study of the atlanto-occipital region requires precise lateral roentgenograms, without rotation of the head. Any movement modifies the relationships and obscures visualization of the region. The study is completed by using other projections and laminagraphy, which is indispensable and which, in selected cases,

* Chief of the Department of Radiology, Neurological Institute, Milano, Italy.

includes the axial transverse plane. This latter method of examination has proved to be of considerable value for the study of the occipital foramen, since it shows its exact shape and actual dimensions free from the frequently observed distortions and superimpositions brought about by projection factors (Fig. 1, *a* and *b*).

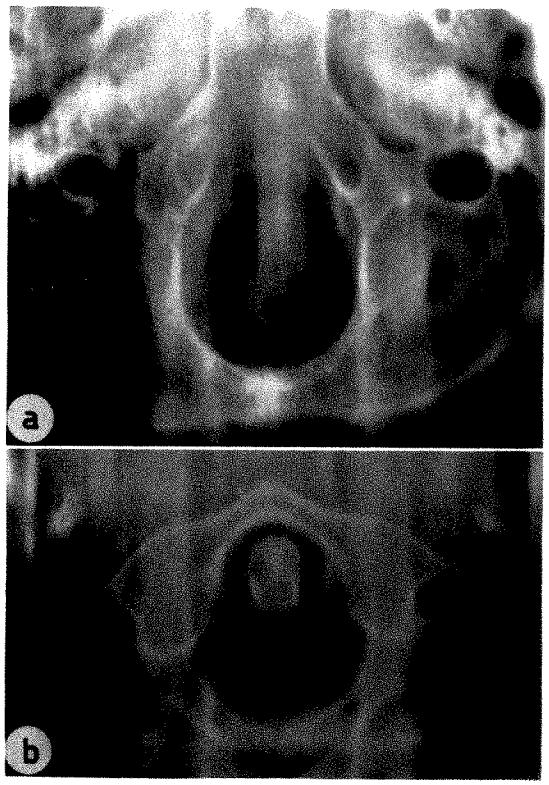


Fig. 1. Axial transverse laminagrams. Sections through (*a*) foramen magnum and (*b*) the atlas.

ROENTGEN FEATURES

THIRD CONDYLE

Early in the last century, Meckel described the third condyle as a median bone formation of exocranial development which detaches itself from the anterior contour of the occipital foramen. The condyle is usually narrow at its base, becoming broader at the level of its inferior surface which bears an articular surface for the apex of the odontoid of the axis or, more rarely, the anterior arch of the atlas. It varies in size; when it shows a pronounced development it can also be observed on plain lateral roentgenograms of the skull (Fig. 2*a*). Hadley[7] mentions specimens 13 and 14 mm. in length and, in the case published by Kamieth,[9] the formation was even bigger. However, as a rule, it is rather small and must be searched for with the aid of laminagraphy (Fig. 2*a'*; and 3, *a-d*). Continuity of the condyle with the basiocipital bone is sometimes indicated by an

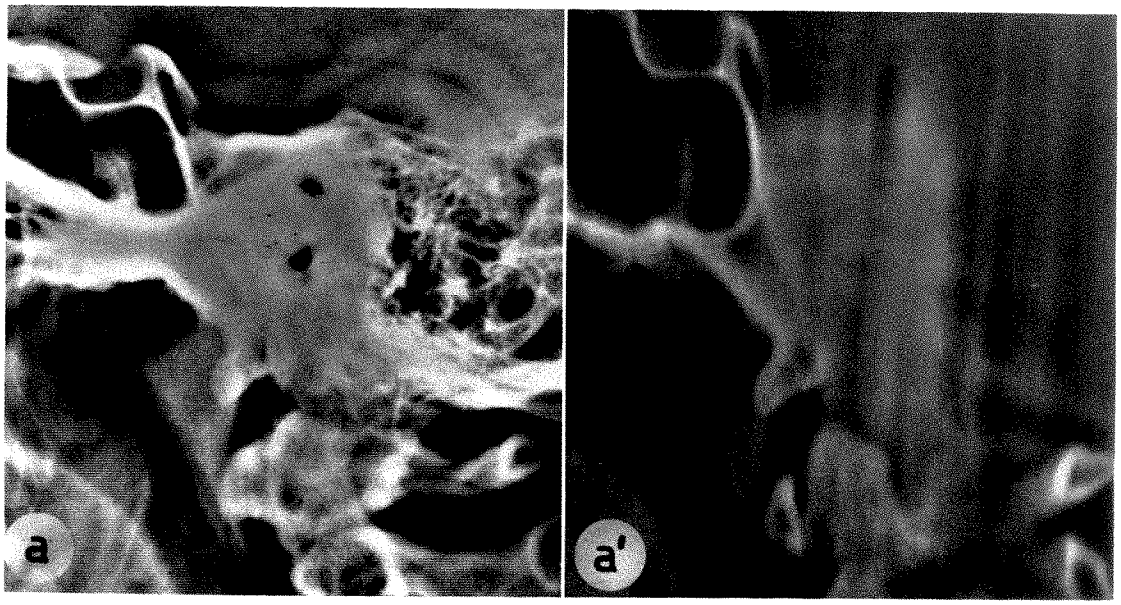


Fig. 2. The third condyle seen on (*a*) a roentgenogram and (*a'*) a laminagram.

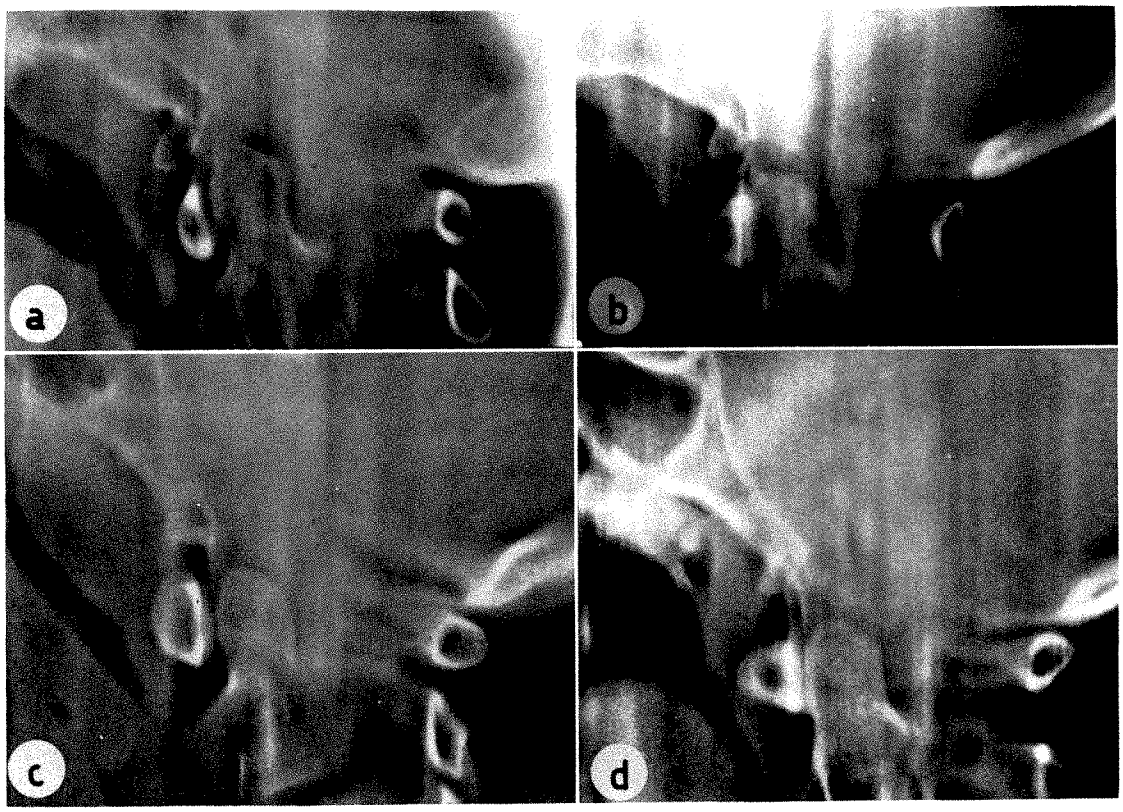


FIG. 3. (*a–d*) Different aspects of the third condyle.

incisura and by a transverse streak of denser bone (Fig. 3, *a* and *b*). The 6 cases we have collected make this the most frequent malformation in our series of manifestations of the occipital vertebra. In 5 cases the anomaly was present in its pure form; in the sixth it was associated with an anomaly of the axis (separate odontoid process). None of our patients had any complaints attributable to the condyle, but it is known that when it is large it may restrict head movements.

According to Kollmann, whose interpretation we will further refer to below, the third condyle is the result of incomplete regression of the hypochordal arch of the occipital vertebra or pro-atlas. Brocher[1] believes that it corresponds to the body of the occipital vertebra as does the odontoid process to the body of the atlas.

PARACONDYLOID PROCESS

In the skull of the adult man, on each side of the occipital condyles, we usually find ridges and rough spots in the bone where muscles or tendons are inserted. When these formations, which embryologically derive from the inferior articular apophysis of the occipital vertebra, are larger than normal, they are termed the "paracondyloid process" or "manifestation of the transverse process of the atlas."[15]

In the works of MacRae and Barnum,[13,14] Franceschetti *et al.*,[6] Epstein,[4] and Kvasnička,[10] the paracondyloid process is confused with the paramastoid process and indicated as such, as if the two terms were synonymous and the anomalies identical. Actually, the anatomic studies of Hayek[8] have shown that the paracondyloid process is a variation which is observed in man, projecting from the paracondyloid surface of the occipital bone and extending in the direction of the transverse process of the atlas, whereas the paramastoid process is a variation observed in many animal species and its usual location is at the posterior border of the jugular foramen. The para-



Fig. 4. A bilateral paracondyloid process.

condyloid process has been compared to the sacralized transverse apophysis of the last lumbar vertebra and, as the latter, it may be uni- or bilateral and may show symmetric or asymmetric development (Fig. 4 and 5*a*). That it orginates from the occipital bone (exo-occipital) is also proved by the presence of pneumatic cells.[1] Tischendorf[20] from an anatomic point of view and Schmidt[16] from a roentgenologic point of view have described a paracondyloid suture situated between the base of the malformation and the occipital bone.

In our material there have been 5 cases of the paracondyloid process—3 unilateral and 2 bilateral. In only 1 case was the malformation present in its pure form; in 3 cases it was associated with assimilation of the atlas and in the remaining case with complex anomalies of the occipitovertebral joint. In 1 case there was an osseous bridge with the atlas, and the paracondyloid suture was never observed. When the paracondyloid process is not fused with the underlying vertebra, it has the shape of a cone, sometimes large, projecting from the occipital bone and with a wide base (Fig. 4). It is bordered by the cortical layer and, in the center, the osseous structure is that of spongy bone; in 1 case pneumatic cells were present (Fig. 5*a*), a phenomenon that appears to be rather rare.[17]

Four patients showed neurologic signs and symptoms that could be attributed to the associated malformations, however, and not to the paracondyloid process

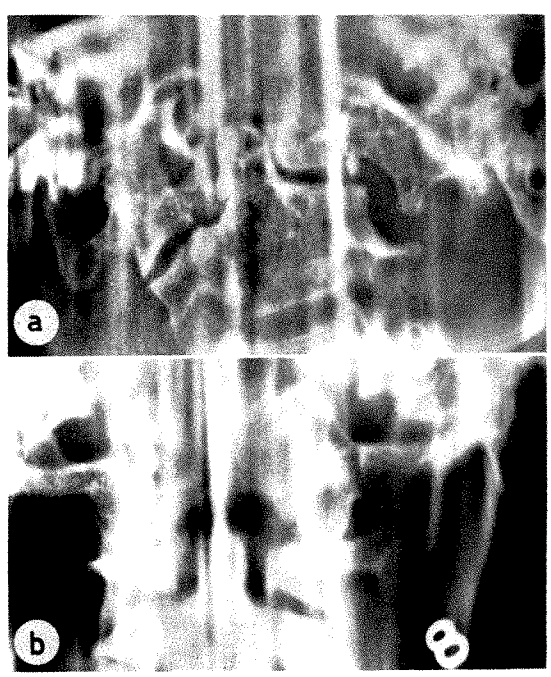


Fig. 5. (*a*) Paracondyloid process on the left. (*b*) Epitransverse process of the atlas fused to the occipital bone.

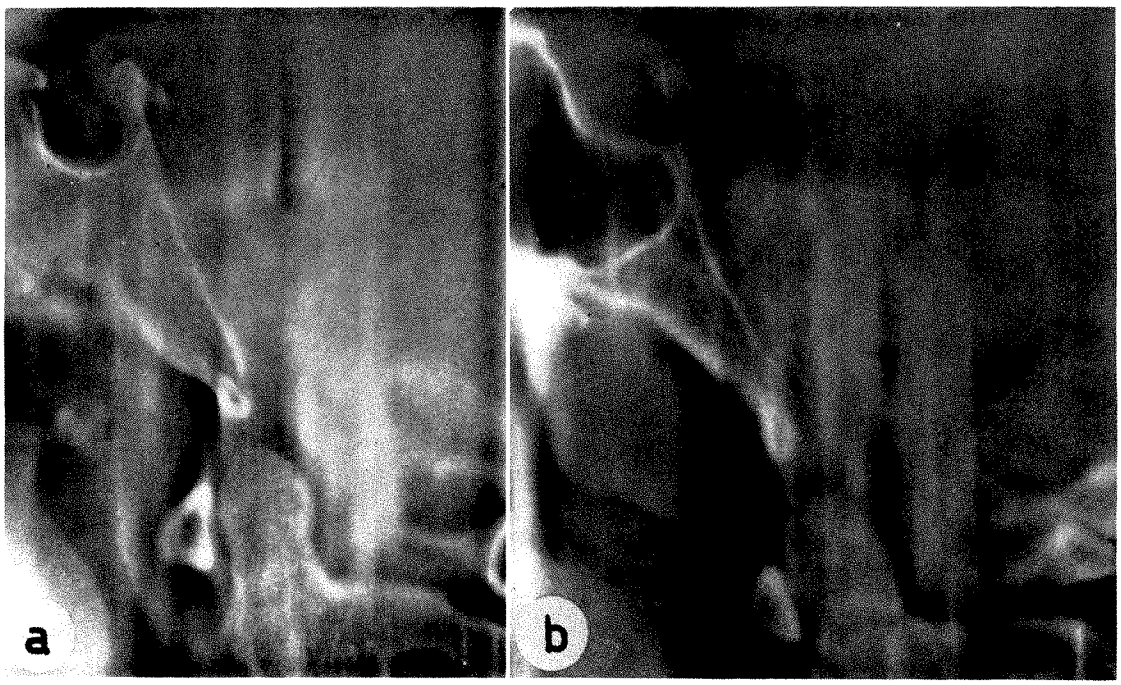


Fig. 6. (*a* and *b*) Basilar processes.

which, in spite of its size and the fusion with the atlas in the fifth patient, caused no disturbances. In Kvasnička's[10] case, on the other hand, the head was fixed in an attitude of flexion.

The paracondyloid process must not be confused with the epitransverse process of the atlas, which is its mirror image (Fig. 5*b*). It is an osseous formation which projects from the transverse process of the atlas with an arch medially concave and extends towards the occipital bone with which it is sometimes fused.[11] The few cases in the roentgenologic literature have been reported by Brocher,[1,2] Viehweger,[22] Schmidt,[16] and Schmidt and Fischer.[17]

BASILAR PROCESSES

Incomplete regression of the lateral parts of the hypochordal anlage may result in the presence of two small, often asymmetric, bone formations disposed along the median line on the inferior surface of the base of the skull at the anterior border of the occipital foramen. Sometimes these formations are in contact with each other, forming a small central canal and simulating a third condyle. Roentgen demonstration of the basilar processes is difficult and they cannot always be recognized with certainty. We were able to diagnose the 2 cases shown in Figure 6, *a* and *b* because of their localization and similarity to the basilar processes seen in the photographs of the dried skull.

ACCESSORY BONE ELEMENTS, SEPARATE OR FUSED TO THE FORAMEN MAGNUM

According to Schumacher,[18] we must regard as a manifestation of the occipital vertebra its ossified fragments which, when imbedded in the ligamentous tissue, appear on roentgenographic examination as isolated foci between the atlas and the occipital bone. We have collected 4 cases of this type and others have been published by Fischer.[5] In the latter's view, the separate bone elements are only partly attributable to the occipital vertebra, because findings due to secondary ossification in the ligaments have been observed.

These accessory formations have the appearance of sesamoid bone and are mostly encountered above the anterior

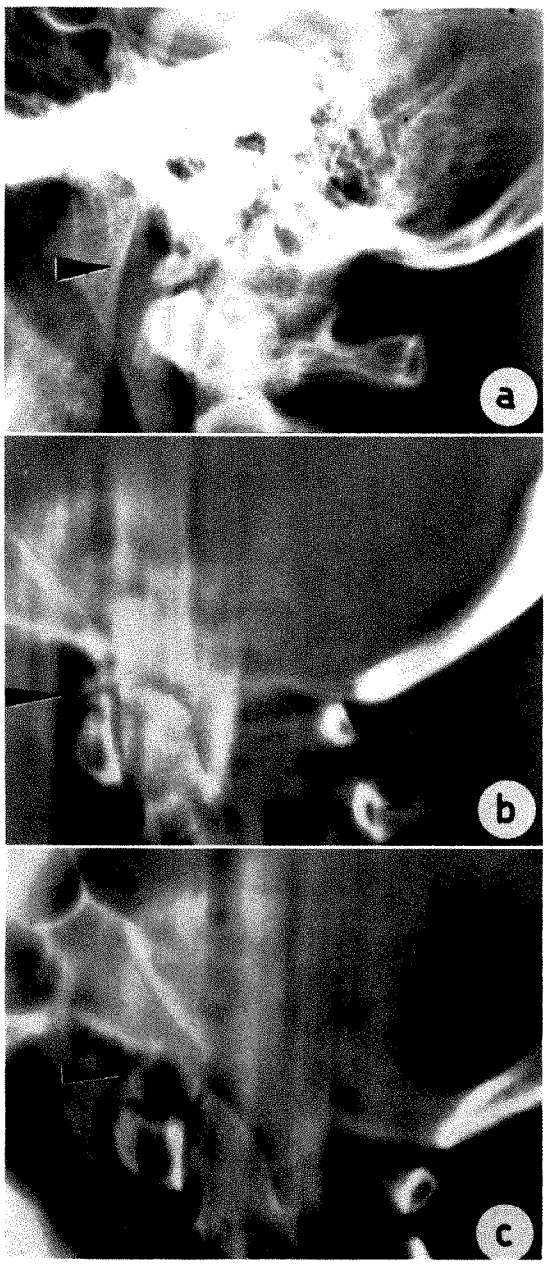


Fig. 7. (*a*, *b* and *c*) Different aspects of accessory bone elements.

arch of the atlas (Fig. 7, *a*, *b* and *c*). This localization renders them easily distinguishable from the ossiculum terminale of Bergmann, a normal anatomic formation which, though not always, is situated on the odontoid process, constituting its tip. In 1 case only, the ossification was localized over the odontoid of the axis, but its semilunar shape clearly pointed to its having originated from a hypochordal arch (Fig. 8). Neither in this patient nor in the other 3 cases were there any physical disturbances ascribable to the malformation.

The neural arch or a transverse process of the pro-atlas may become fused, wholly or in part, with the lower rim of the occipital foramen, which is pinched in and deformed by osseous formations (labia foraminis magni). The case shown in Figure 9 is an example. The patient, a fifty-one year old male, had noted five years previously the appearance of a spastic paraparesis of the lower extremities which progressed slowly and which, after four years, involved the upper extremities as well. When the patient was admitted to the hospital he presented a spastic tetraparesis, more marked in the lower extremities, with mild hypotrophy of the scapulohumeral musculature and slight bilateral superficial hypoesthesia from C3 to C6. Roentgen examination revealed a fairly complicated malformation. In addi-

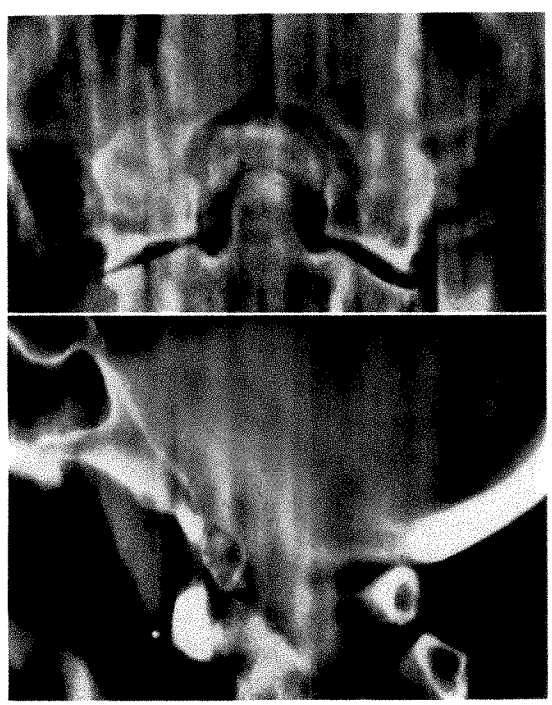


Fig. 8. Accessory bone on the anterior part of the foramen magnum.

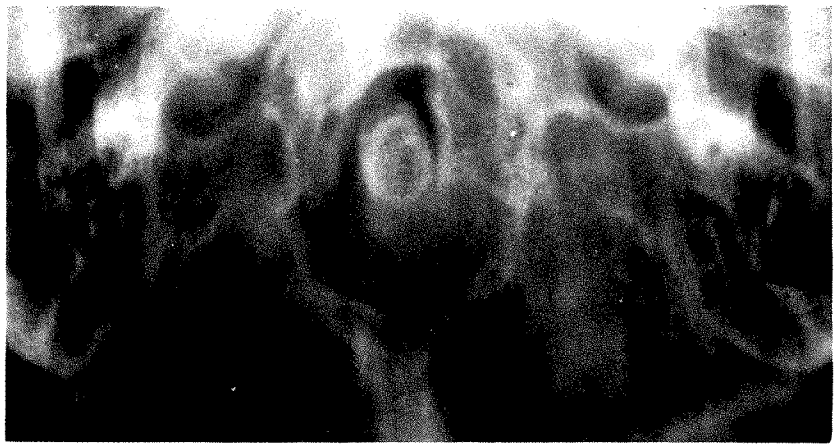


FIG. 9. An axial transverse laminagram showing distortion of the foramen magnum by an anterolateral bony mass on the left and by posterior luxation of the odontoid process.

tion to the asymmetric basilar impression with platybasia and posterior dislocation of the odontoid process of the axis, two simultaneous varieties of manifestation of the occipital vertebra were present, *viz.*, a paracondyloid process and a bony mass distorting the occipital foramen (Fig. 9). In view of the complex character of the malformation encountered, it was impossible to determine the part each of these played in provoking the clinical symptomatology exhibited by the patient. In bilateral cases, *e.g.*, the case reported by Hadley,[7] the occipital foramen showed a deformation in the shape of a bicycle saddle.

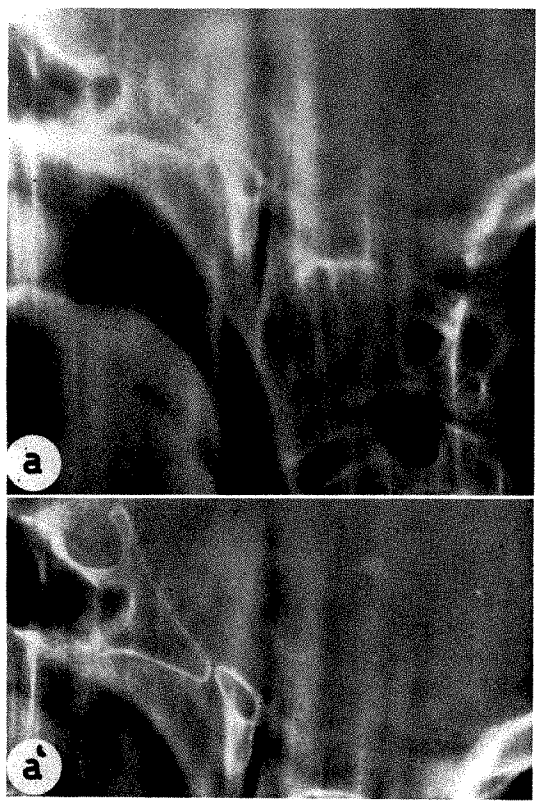


FIG. 10. (*a*) Laminagram with paramedian section showing lateral segmentation of the basiocciput. This fissure is of the same type as 1 in Figure 11. (*a'*) A retouched reproduction from *a*.

TRANSVERSE FISSURES OF THE BASILAR PROCESS OF THE OCCIPITAL BONE

These constitute another aspect of manifestation of the occipital vertebra. Very rarely, the fissure may be complete, in which case it borders an arch which Chiarugi has called the "pre-basioccipital arch." More often, it is partial, uni- or bilateral.[11,15–17] Neither variety has clinical significance. As far as we could determine, roentgenologic documentation is available only for a case described by Schmidt and Fischer[16,17] and one by List[12] which showed a unilateral fissure of the basiocciput and assimilation of the atlas. Our case (Fig. 10, *a* and *a'*; and 12*c*) is identical with the latter even as regards the associated malformation. Laminagraphy of the basiocciput performed in a lateral plane revealed a distinct interruption.

Figure 11 illustrates, by line drawings

superimposed on a laminagram, the various types of fissures observed in photographs of anatomic specimens.

DISCUSSION

In contrast to previous assumptions, and with a frequency surpassing that mentioned in the literature, it has been our experience that roentgenologic investigations may reveal the bone anomalies of the manifestation of the occipital vertebra.

These are anomalies of considerable interest anatomically and, from the phylogenic point of view, they have the significance of a regressive trait. There is an apparent contradiction, explained by embryology, in that they have morphologic characteristics of a vertebral type identical to those of the atlas, although they belong to the skull (spondylocranium), as is clearly evidenced by their fusion and assimilation with the occipital bone.

These malformations are rare. Sauser[15] has observed only 5 cases among 1,119 skulls in an ossuary (0.44 per cent); we have found 19 such cases in 4,000 patients (0.47 per cent). The relative frequency of our observations is, therefore, the same as that of Sauser,[15] which can be explained by the fact that the limitations of a roentgen examination are offset by the demonstration of the isolated ossifications, which become lost in the dried skull.

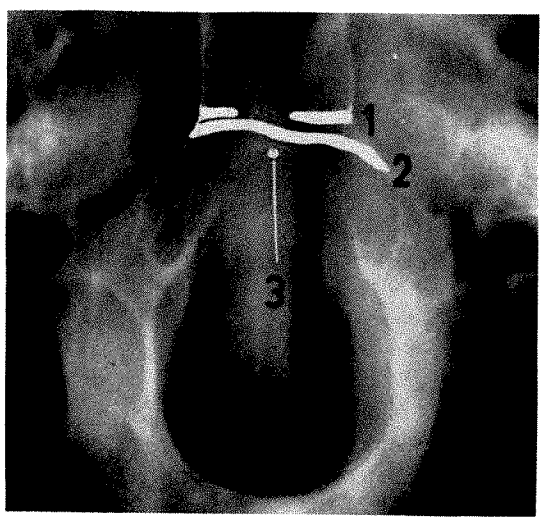


Fig. 11. Types of fissures schematically indicated by superimposed lines: (1) lateral segmentation, (2) complete segmentation of the basiocciput due to lack of assimilation of the most hypoglossal sclerotome, (3) pre-basioccipital arch.

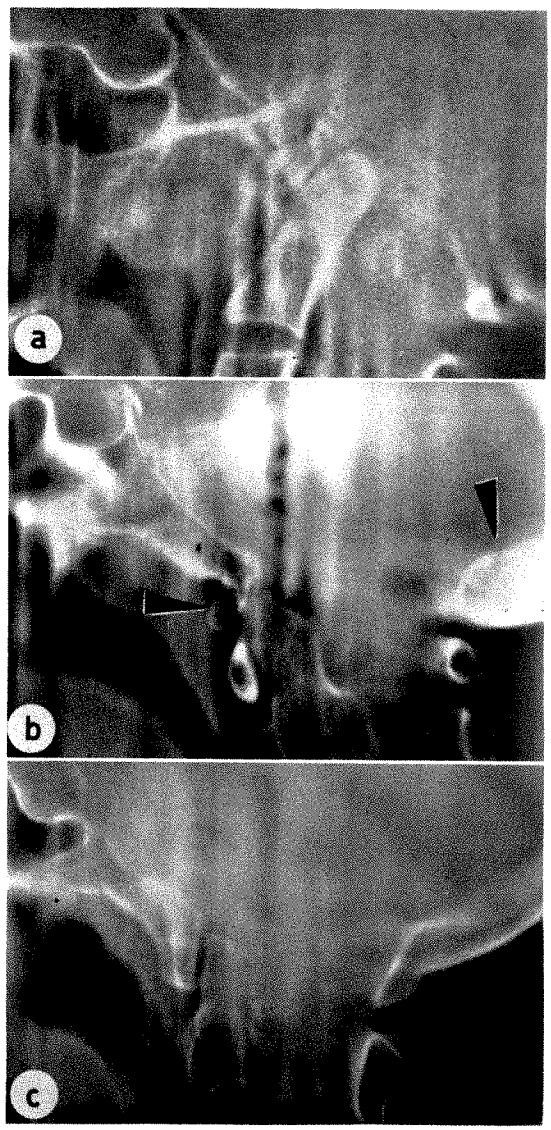


Fig. 12. Laminagrams demonstrating (*a*) dysplasia of the basiocciput, (*b*) manifestation of the occipital vertebra with deformity of the anterior and posterior parts of the foramen magnum, and (*c*) atlanto-occipital fusion with the second and third cervical block vertebrae. (Same case as in Figure 10, *a* and *a'*.)

Most of the time manifestations of the occipital vertebra have no clinical significance. There are rare exceptions: the third condyle, when well-developed, may limit

the mobility of the head; the paracondyloid process, fusing with the transverse process of the atlas, may force the head into a continuous attitude of flexion (caput obstipum) and, most importantly, the abnormal osseous formations which originate from the arches or from the transverse apophyses of the pro-atlas, when fused with the base, will narrow the occipital foramen and may provoke neurologic symptomatology of a compressive type.

The roentgen demonstration of these particular cases is exceptional, but it is to be expected that more cases will be diagnosed in the future, mainly as the result of the application of axial transverse laminagraphy. At the present time, so far as we have been able to ascertain, the only cases that have been diagnosed in the living patient have been the one reported by Hadley[7] (in which operation was carried out), the case of Brocher and Klein[3] (twelve year old boy who presented an incomplete form of mandibulofacial dysostosis [syndrome of Treacher-Collins]), and the case reported in the present communication.

The manifestation of the occipital vertebra may be present in its pure form or in association with other local malformations. In our group of cases, the pure form was encountered more often (11 cases) than the associated form (8 cases). In this connection we wish to call attention to the high frequency of the paracondyloid process in cases of assimilation of the atlas. The 5 cases out of 25 reported by McRae and Barnum[14] show the same proportional frequency as the 3 out of 14 cases in our material; the causes of this curious association are not clear.

One patient, in addition to malformations of another nature, presented a paracondyloid process and a bony mass distorting the occipital foramen, *viz.*, two variations of manifestation of the occipital vertebra.

The study of this malformation has, furthermore, demonstrated the possibility of reconstructing roentgenologically even to details, the complex evolutive developments of the occipitovertebral region and of compiling a sequentially progressive list of malformations: severe dysplasia of the basiocciput, manifestation of the occipital vertebra, and assimilation of the atlas (Fig. 12, *a*, *b* and *c*). The links between these stages are intermediate forms.

CONCLUSIONS

The occipital vertebra is a rare malformation which, although known to anatomists, is not very familiar to radiologists.

Though the anomaly is normally asymptomatic, it may, alone or in combination with other local malformations, provide neurologic signs and symptoms.

Roentgen examination of the craniovertebral junction in a consecutive series of 4,000 patients has revealed manifestation of the occipital vertebra in 19 cases (0.47 per cent).

The most common of these malformations is the third condyle, followed in descending order of frequency by the paracondyloid process, isolated ossification between the atlas and the occipital bone, basilar processes, fissures of the basiocciput, and a bony mass fused with the occipital foramen. The last mentioned variant appears to be the most dangerous form because it may lead to direct compression of the spinal cord.

Neurological Institute
Department of Radiology
11 Via Celoria
Milano, Italy

REFERENCES

1. BROCHER, J. E. W. Die Occipito-Cervical-Gegend. Georg Thieme, Stuttgart, 1955.
2. BROCHER, J. E. W. Neuere Ergebnisse der Wirbelsäulendiagnostik. Schinz, H. R., Glauner, R., and Uehlinger, E. Röntgendiagnostik. Ergebnisse 1952–1956, pp. 56–72. Georg Thieme, Stuttgart, 1957.
3. BROCHER, J. E. W., and KLEIN, D. Die Dysostosis mandibulofacialis im Röntgenbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1960, *93*, 67–79.
4. EPSTEIN, B. S. The Spine: A Radiological Text and Atlas. Lea & Febiger, Philadelphia, 1955.
5. FISCHER, E. Akzessorische freie Knochenele-

mente in der Umgebung des Foramen occipitale magnum. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1959, *91*, 638–642.

6. FRANCESCHETTI, A., BROCHER, J. E. W., and KLEIN, D. Dysostose mandibulofaciale unilatérale avec déformations multiples du squelette et torticollis cronique. *Arch. Klaus Stiftung Vererbungforsch.*, 1947, *22*, 373. Quoted by Schmidt.
7. HADLEY, L. A. Atlanto-occipital fusion, ossiculum terminale and occipital vertebra as related to basilar impression with neurological symptoms. AM. J. ROENTGENOL. & RAD. THERAPY, 1948, *59*, 511–524.
8. HAYEK, G. V. Quoted by Schmidt and Fischer.[17]
9. KAMIETH, H. Ein nicht sicher hinzuordnender Knochenkeil am Unterrand des Clivus. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1959, *91*, 334–339.
10. KVASNIČKA, I. Processus paramastoideus als Ursache einer schiefen Kopfhaltung. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1958, *88*, 744–746.
11. LE DOUBLE, A. F. Traité des variations des os du crâne de l'homme. Vigot Frères, Paris, 1903.
12. LIST, C. F. Neurological syndromes accompanying developmental anomalies of occipital bone, atlas and axis. *Arch. Neurol. & Psychiat.*, 1941, *45*, 577–616.
13. MCRAE, D. L. Bony abnormalities in region of foramen magnum: correlation of anatomic and neurologic findings. *Acta radiol.*, 1953, *40*, 335–355.
14. MCRAE, D. L., and BARNUM, A. S. Occipitalization of atlas. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1953, *70*, 23–46.
15. SAUSER, G. Manifestation der Okcipitalplatte an einem menschlichen Schädel. *Ztschr. Anat. u. Entw. Gesch.*, 1933, *102*, 51–67.
16. SCHMIDT, H. Okzipitale Dysplasien. Die Manifestation des Okzipitalwirbels im Röntgenbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1959, *90*, 691–704.
17. SCHMIDT, H., and FISCHER, E. Die Okzipitale Dysplasie. Georg Thieme, Stuttgart, 1960.
18. SCHUMACHER, S. Ein Beitrag zur Manifestation des Okcipitalwirbels. *Anat. Anz.*, 1907, *31*, 145. Quoted by Brocher.[2]
19. STEVENSON, J. J. Horizontal body section radiography. *Brit. J. Radiol.*, 1950, *23*, 319–334.
20. TISCHENDORF, F. *Ztschr. Morph. Anthrop.*, 1952, *43*, 361. Quoted by Schmidt.[16]
21. VALLEBONA, A. Axial transverse laminagraphy. *Radiology*, 1950, *55*, 271–273.
22. VIEHWEGER, G. Der Prozessus paratransversarius, eine Varietät am Atlas. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedzin*, 1955, *83*, 411–412.

ANATOMICO-ROENTGENOGRAPHIC STUDIES OF THE POSTERIOR SPINAL ARTICULATIONS

By LEE A. HADLEY, M.D.
SYRACUSE, NEW YORK

KELLER,[2,3] from his studies of the apophyseal articulations, has reported that local back pain arises from the posterior spinal joints. The present study was undertaken for the purpose of coordinating the roentgenographic appearance with the anatomic condition of these structures.

ANATOMY AND FUNCTION

The lower element of the joint, *i.e.*, the superior articular process, is concave or partially cylindrical in the lumbar region, while the inferior articular process is biconcave (Fig. 2). Since the surfaces are not completely in contact, meniscus-like tabs of synovial tissue project inward from the joint margins. These lie partially between the articular surfaces. Some of them are thick and fleshy; others are thin, fibrous and ragged. They are made up of fibrous tissue, fibrocartilage, fat cells and many blood vessels (Fig. 2, 5 and 6).

Medially and anteriorly, the joint capsule is formed by a lateral continuation of the tough ligamentum flavum. Posteriorly, the capsule is much thinner and is loosely attached, not to the margins of the joints, but reflected around to the outer surfaces of the articular processes. The articular cartilage likewise may extend well beyond the limits of bony contact. The joint space

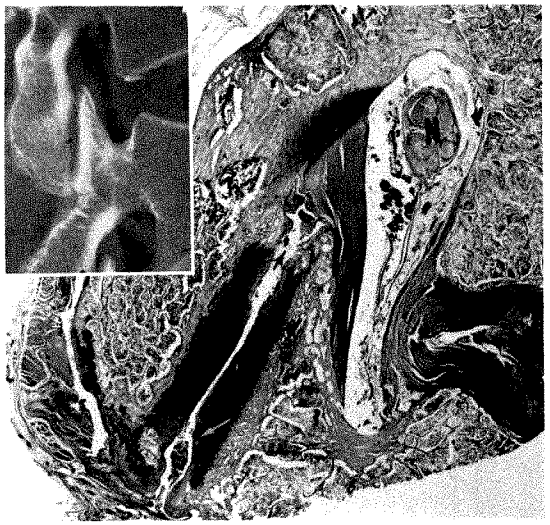


FIG. 1. Sagittal section (×2) of a normal fourth posterior lumbar articulation in a thirty-seven year old male. The capsule above, formed from the ligamentum flavum, separates the joint from the normal sized intervertebral foramen on the right. Note that the diameter of the nerve (N), which in the lumbar region occupies the upper portion of the foramen, is no more than one-fifth the size of the opening which normally transmits it. The dark articular cartilage on both sides shows a normal thickness. Tabs of synovial tissue are visualized above and below (arrows). Note the customary weakness of the capsule at the bottom. (The insert is a roentgenogram of this joint.)

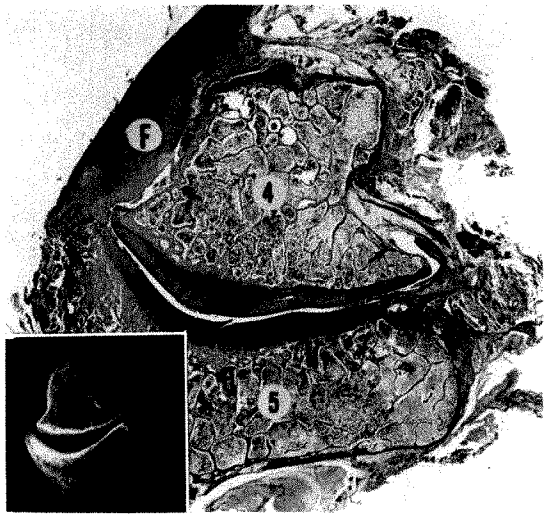


FIG. 2. Transverse section (×3) of the fourth posterior articulation in an eighty-one year old male. The articular surface of the lower element from the fifth vertebra inferiorly and anteriorly is concave and covered with hyaline cartilage (5). This opposes the convex cartilaginous surface of the upper element (4). The ligamentum flavum (F) constitutes the capsule on the medial aspect of the joint. The arrows indicate the reflection of the capsule from the bone on each side. Note the laxity of the capsule and the synovial tissue lying within it. (The insert is a roentgenogram of the specimen before section.)

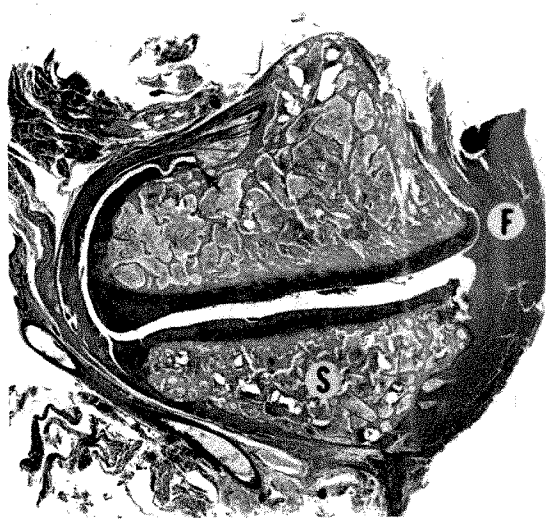


Fig. 3. Transverse section (×3) of the fifth posterior articulation from a seventy-three year old male. At the top is the triangular section from the tip of the fifth inferior articular process; the articular process from the sacrum is visualized below (S). A cushion of cartilage is reflected around the right articular margin sustaining the sidewise thrust of the joint by its pressure against the ligamentum flavum at F, which here constitutes the anteromedial portion of the capsule. On the left, the side thrust is opposed by the inside of the capsule where the cartilage is reflected completely around to the posterior surface of the articular process (arrow). It will be seen that in the lower lumbar region the posterior joint offers considerable laxity of movement in all directions and that the cartilage articulates with ligamentous structures.

may thus be actually extended well around to the posterior surface of the articular process. Laxity of the capsule posteriorly and inferiorly allows considerable range of movement in different directions. The cushions of cartilage along the sides of the articular processes serve as bearing points against the inside of the capsule of the joint. This enables it to resist the sideways thrust of the upper element of the articulation (Fig. 3 and 4).

From a study of cadaver material as well as routine roentgenograms, it is obvious that these joints permit various types of gliding movement. Upon anterior flexion the upper element glides upward and forward upon the lower element, the articular surfaces separate at the lower margins of the joint and the foramen opening becomes larger. Only a portion of the articular surfaces may remain in contact. Upon backward bending the joint surfaces telescope into each other and the foramina become smaller. Upon rotation, the articular sur-



Fig. 4. Dissected fifth lumbar capsule from a seventy-three year old male showing how the structure is attached well beyond the articular margin. The synovial tab which projects between the joint surfaces like a meniscus also can be seen. The overhang of the joint margin is much greater at the lumbosacral level than it is above. On the opposite side, this subject presented a double fat pad with smooth surfaces which acted as a cushion between the tip of the fifth right inferior articular process and the neural arch of the sacrum.



Fig. 5. The third right posterior articulation from a sixty-nine year old male laid open to show the articular surfaces and the synovial menisci. The capsule was detached from the posterior surface of the upper element at X and was retracted downward. It bears a long, thin, ragged, fibrous synovial tab or meniscus (black arrows). A thick fleshy meniscus of fatty tissue is seen attached to the upper portion of the capsule but displaced slightly downward (white arrow). The thick meniscus was present above because the upper margins of the joint were well separated. The darker, concave, lower element of the joint beneath the fat pad was roughened by early degenerative change. Note also some roughening of the posterior edge of the upper element just to the right of X.

faces press together on one side and tend to pull apart on the other. With lateral flexion, balanced gliding movements occur with the upper joint margins separating on the convex side of the curve and imbrication or telescoping occurring on the concave side.

Sections of the joint capsule studied by special staining methods visualized a rich supply of sensory nerves (Fig. 7). May we not assume the possibility of a pinching of the soft tissue structures of the joint as a cause of the "sudden catch" type of low back pain? Clinically, this is characterized by the patient's inability to completely straighten up after bending forward.

DEGENERATION

Normally, the articular surfaces are covered by smooth glistening hyaline



Fig. 6. Photomicrograph (×4) showing a large synovial tab (arrow) projecting upward between the articular surfaces. This originates from the inferior, loose portion of the capsule. The structure in this instance is largely composed of fatty tissue. The articular cartilage on the right side is partially worn down but the hyaline cartilage is of normal thickness on the left. This synovial tab might conceivably become pinched between the bony structures.

cartilage. Early degenerative changes first appear as a velvety texture overlying the articular surface (Fig. 5). Later, the cartilage becomes fibrillated and fragmented and finally disappears entirely in some parts of the joint. Roentgenograms of these joints resemble those of some patients complaining of local back pain (Fig. 8).

IMBRICATION, IMPINGEMENT, BUMPER FORMATION, EBURNATION AND EROSION

Lordosis, tipping of the sacrum, or thinning of the intervertebral disk results in a telescoping or imbrication of the posterior articulations (Fig. 9, *A* and *B*). The articular surfaces are no longer exactly opposite each other. A pulling stress is placed upon the capsule. The anterior inferior element of the joint, by moving upward and forward, may encroach upon the foramen, even to the extent of pressing upon and distorting the shape of the nerve root.

If imbrication is excessive, a painful bony impingement may occur between the tips of the articular processes and the adjacent portions of the neural arch (Fig. 10). A fibrocartilage pad or bumper develops on the tip of the impinging articular process at the point of recurrent pressure (Fig. 11, *A* and *B*) and a sclerosis or eburnation of the underlying bony structure results (Fig. 12, *A*, *B* and *C*). This tends to counter the localized pressure stress. With continued pressure at one point, however, the condition progresses to actual erosion of the underlying bony tissue.

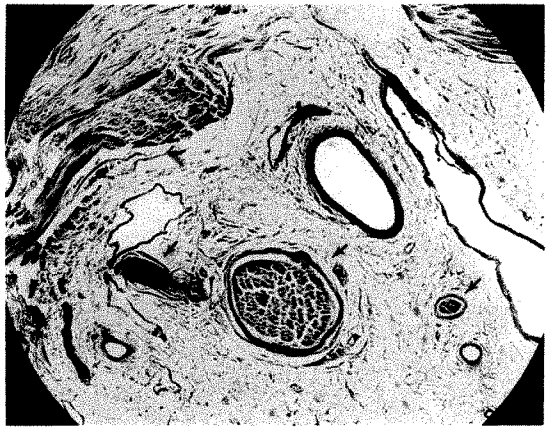


Fig. 7. Section (×30) from the posterior joint capsule showing at least three sensory nerves (arrows). In the center is a large mixed nerve having both myelinated and unmyelinated fibers. Some of the latter are seen branching off from its upper surface. Smaller nerves appear on the right and left. The former is seen in cross section, while the dark oval shadow on the left is an obliquely sectioned sensory nerve. (This section was stained by a modified Bodian and Ungewitter silver nerve stain.)

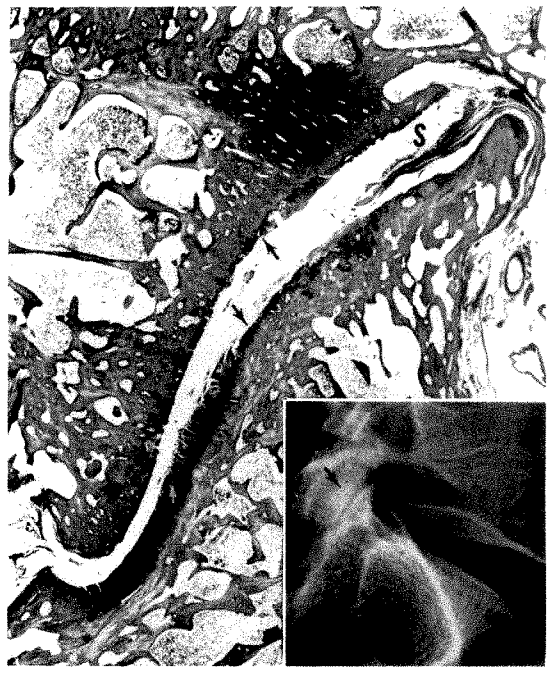


Fig. 8. Longitudinal section (×5) of a degenerated fifth posterior joint from a seventy-five year old male. This shows the synovial element (S) projecting into the upper margin of the joint space. The cartilage is completely absent from the upper element of the joint and there is advanced cartilage degeneration with thinning and fibrillation below (white arrow). (The insert is a roentgenogram of the specimen before section showing the narrowed joint space.) Sclerosis of the bone adjacent to this degenerated joint is well seen both on the section and the roentgenogram (black arrows).

COMMENT

Harris and Macnab[1] have discussed the relationship of symptoms to posterior joint changes. Assuming, as Keller[2,3] has

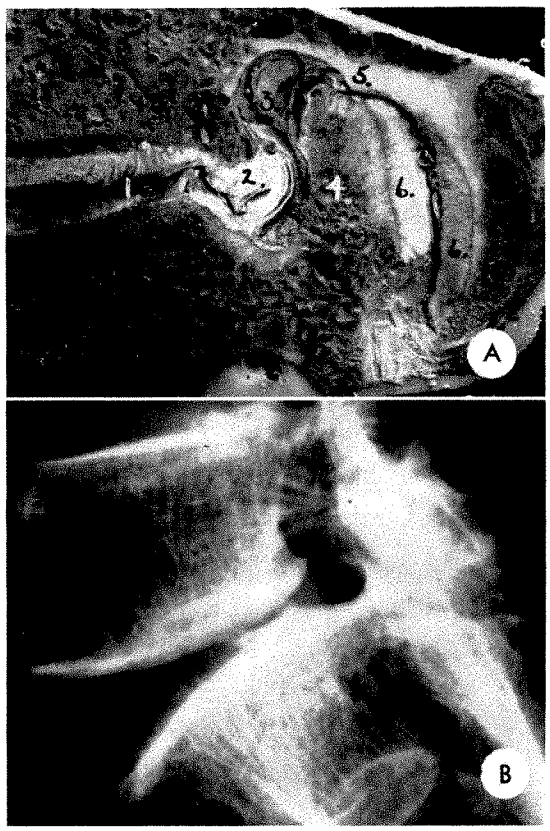


FIG. 9. (*A*) Section at the lumbosacral level from an eighty-six year old male showing imbrication of the posterior articulation. The articular cartilages (6) do not register exactly opposite each other. The articular process of the sacrum (4) has telescoped upward, impinging against the eburnated fifth pedicle at 5 and distorting the nerve root (3). (*B*) Roentgenogram showing the wedge-shaped lumbosacral disk. There is some tipping of the sacrum on a transverse axis. The fifth vertebra is displaced backward upon the sacrum. The disk (1 in *A*) is thinned posteriorly and its annulus fibrosus (2 in *A*) has become displaced backward and encroaches upon the lower part of the intervetebral foramen.



FIG. 10. Section of the fourth right posterior articulation of a seventy-three year old female. The insert is a roentgenogram showing impingement of the tip of the fourth inferior articular process (4) against the isthmus of the fifth neural arch. This has produced erosion with the formation of a compensatory eburnation of bony tissue at the point of pressure (X). The tips of the articular processes subjected to intermittent pressure usually develop fibrocartilage bumpers at points where the normal hyaline articular cartilage does not occur. Such bumpers are noted in the section as indicated by the arrows. Richly vascular synovial tabs are seen above and below at S. There is advanced cartilage degeneration with fibrillation and complete loss of cartilage in the upper portion of the joint.

»»→

FIG. 12. (*A*, *B* and *C*) Scoliosis in an eighty-four year old female incidental to wedging of the lumbosacral disk and the fifth vertebral body (5). There is telescoping, impingement, eburnation and erosion of the fifth posterior articulation on the right side (R, arrow). Compare with the normal condition of its fellow on the left (L).

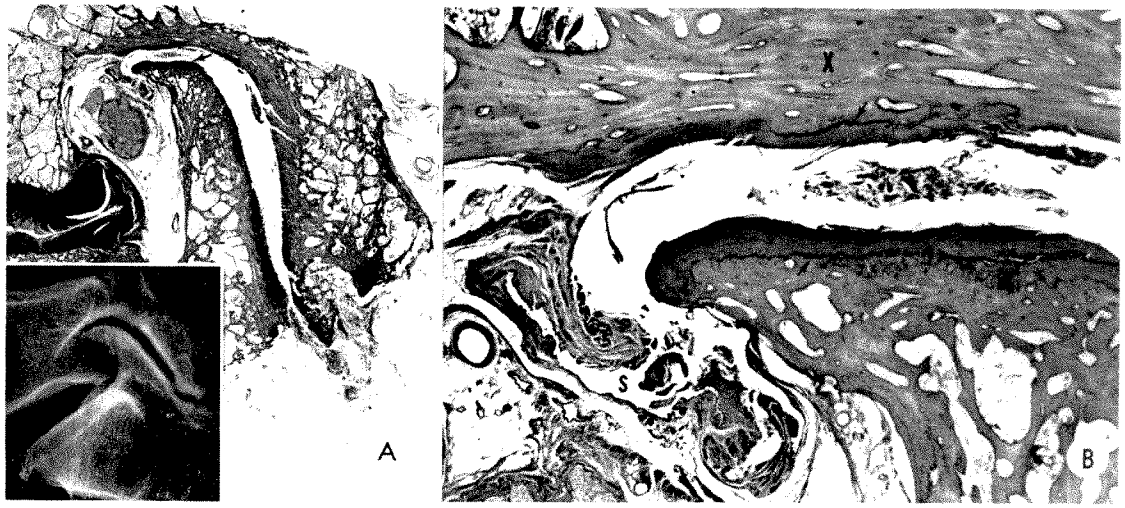
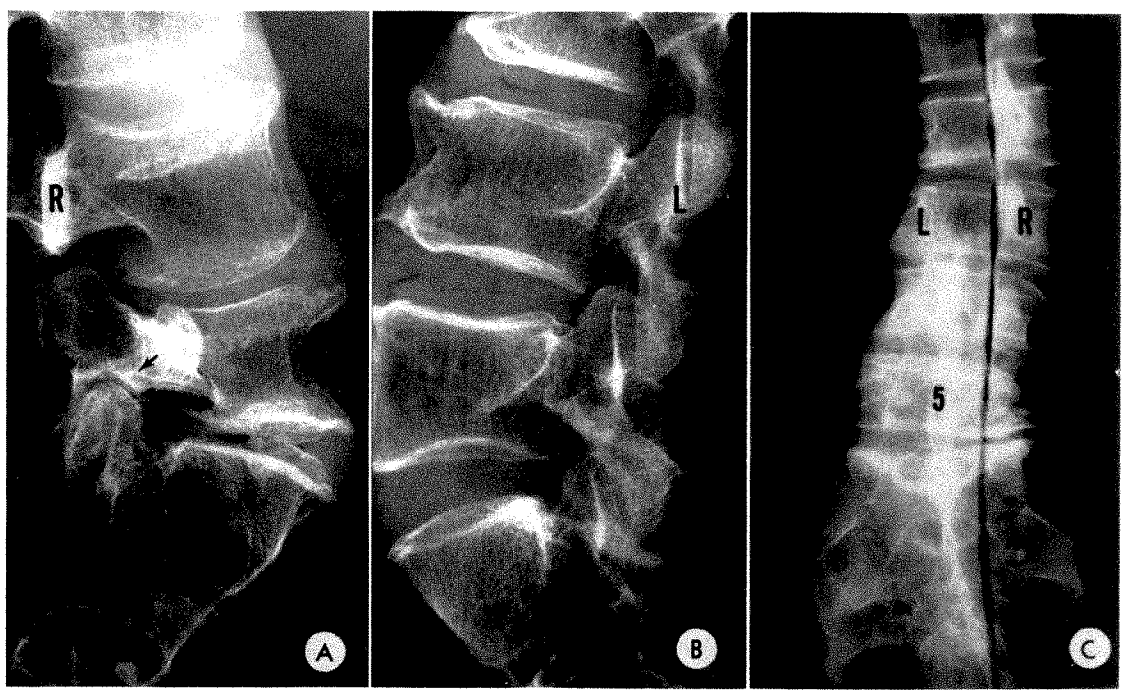


Fig. 11. (*A*) Longitudinal section (×1½) of the fifth lumbar articulation of a seventy-nine year old female. Imbrication and bony spur formation has progressed until extensive impingement has occurred beneath the fifth pedicle with eburnation of the latter. A fibrocartilage bumper at the point of contact has developed on the tip of the sacral articular process (arrow). There is nearly complete loss of articular cartilage below this point. The intervertebral foramen on the left is not involved. Two elements of the motor nerve root are seen slightly above and to the left of the much larger, darker sensory nerve root. (The insert is a roentgenogram showing sclerotic change in the bony structure of the joint.) (*B*) Photomicrograph (×18) of the tip of the sacral articular process showing the fibrocartilage bumper (B), a completely anomalous structure which has developed at the point of pressure. This is not the hyaline type of cartilage which normally covers the articular surfaces, as seen in Figure 6. The opposing sclerotic bone of the pedicle (X) is also protected by a slightly thinner coat of fibrocartilage. These cartilage bumpers are separated from the underlying bone by a narrow zone of darkly staining active cells (arrows). Evidence of new bone growth is seen in the spur formation beneath the bumper and just to the left of the lowermost arrow. Tabs of highly vascularized synovial tissue (S) are noted between the joint and the intervertebral foramen. This section was taken somewhat lateral to the ligamentum flavum, so the capsule between the joint and the intervertebral foramen is thin and delicate as compared with that noted in Figure 1.



stated, that local back pain arises in the posterior joint structures, a coordination of the roentgenograms with the anatomic studies should contribute something toward an understanding of this problem.

SUMMARY

The posterior spinal articulations are gliding joints with a considerable range of movement. There are menisci of synovial tissue projecting inward between the bony articular surfaces. The joint capsule is rich in sensory nerve fibers. The possibility that pinching of the soft tissues causes the "sudden catch" type of low back pain is proposed. Various kinds of joint destruction, with imbrication, impingement, bumper formation, eburnation and even erosion, probably play a part in the production of local back pain. The 45° stereoscopic projection is recommended to best visualize the joints in roentgenography of the lumbar region.

925 State Tower Building
Syracuse, New York

Assistance was given in these studies by the Departments of Anatomy and Photography, New York State School of Medicine, Syracuse, New York.

REFERENCES

1. Harris, R. I., and Macnab, I. Structural changes in lumbar intervertebral discs; their relationship to low back pain and sciatica. *J. Bone & Joint Surg.*, 1954, *36B*, 304–322.
2. Keller, G. Die Bedeutung der Veränderungen an den kleinen Wirbelgelenken als Ursache des lokalen Rückenschmerzes; Auswertung der histologischen Befunde. *Ztschr. Orthop.*, 1953, *83*, 517–547.
3. Keller, G. Die Arthrose der Wirbelgelenke in ihrer Beziehung zum Rückenschmerz (Weitere mikroskopische Befunde an Lendenwirbelgelenken). *Ztschr. Orthop.*, 1959, *91*, 538–550.

A VERTICAL FRACTURE OF THE FIFTH CERVICAL VERTEBRA WITHOUT NEUROLOGIC SYMPTOMS

By CARL M. MANSFIELD, CAPTAIN, USAF, MC
OMAHA, NEBRASKA

VERTICAL fractures of the cervical vertebrae are almost without exception accompanied by quadriplegia or paraplegia.

This article reports a case of a vertical fracture of C_5 with no neurologic symptoms.

REPORT OF A CASE

A twenty-seven year old male was admitted for observation following a history of an automobile accident; he had suffered momentary unconsciousness when his small foreign car had been struck from the rear by a truck.

Physical examination on admission was completely negative, except for a hematoma in the occipital region.

Roentgen examination showed evidence of a vertical fracture of C_5 with a slight compression of the anterior-superior portion. No other abnormality of the cervical spine was noted (Fig. 1, *A* and *B*).

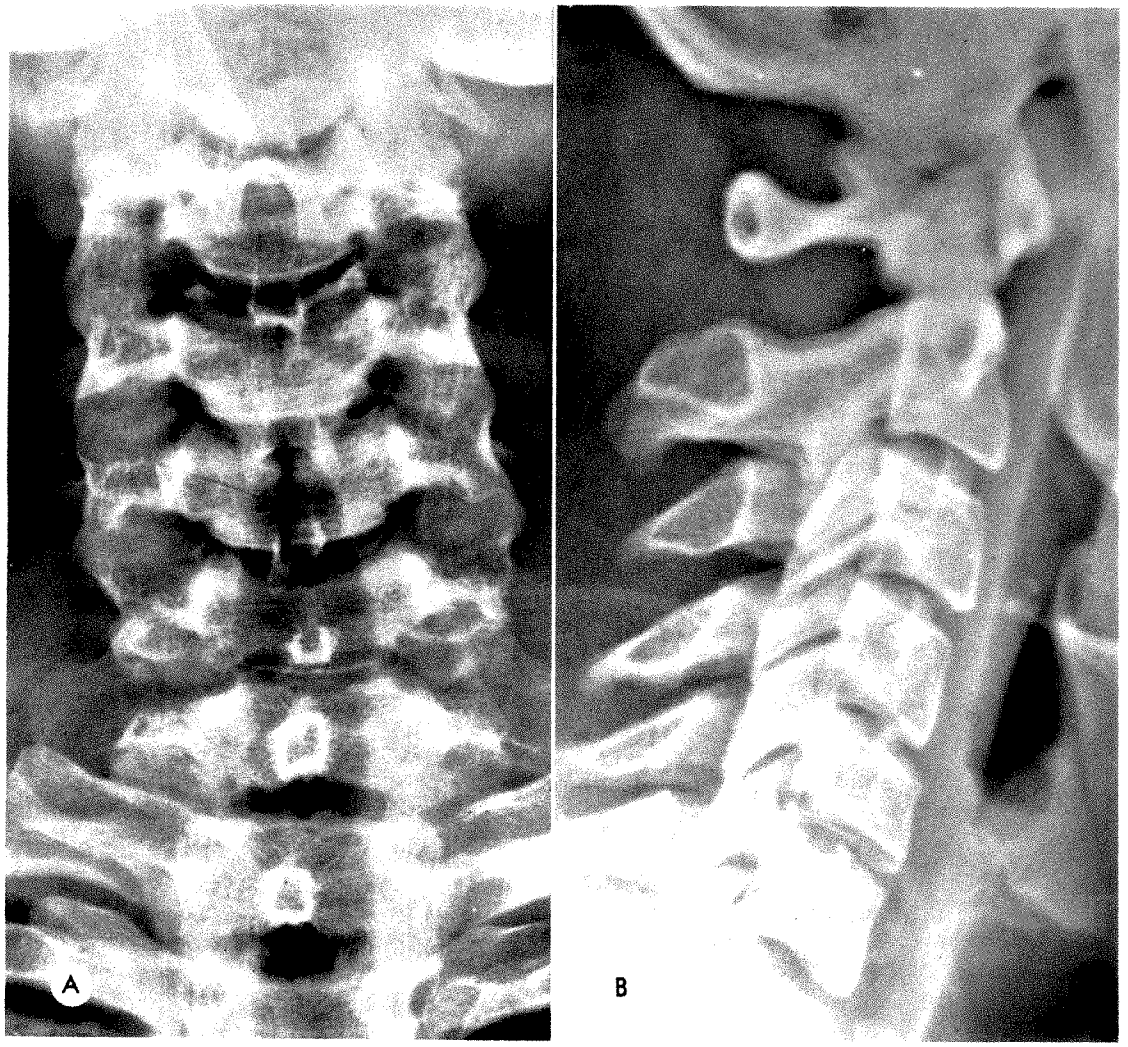
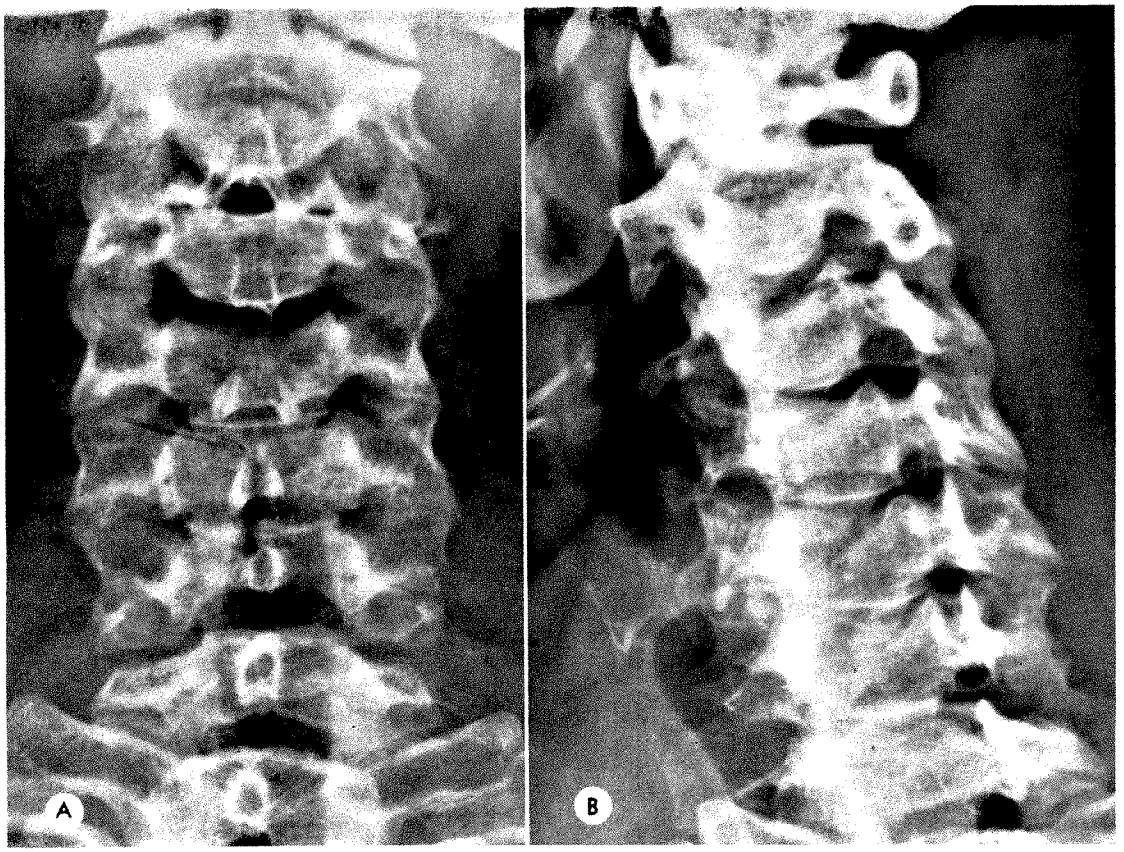


FIG. 1. (*A*) Roentgenogram showing vertical fracture of C_5. Note the downward displacement of the left half of the vertebral body. The radiolucent line is seen within the air column. (*B*) Note that there is some compression of the anterior height of C_5.


A
B

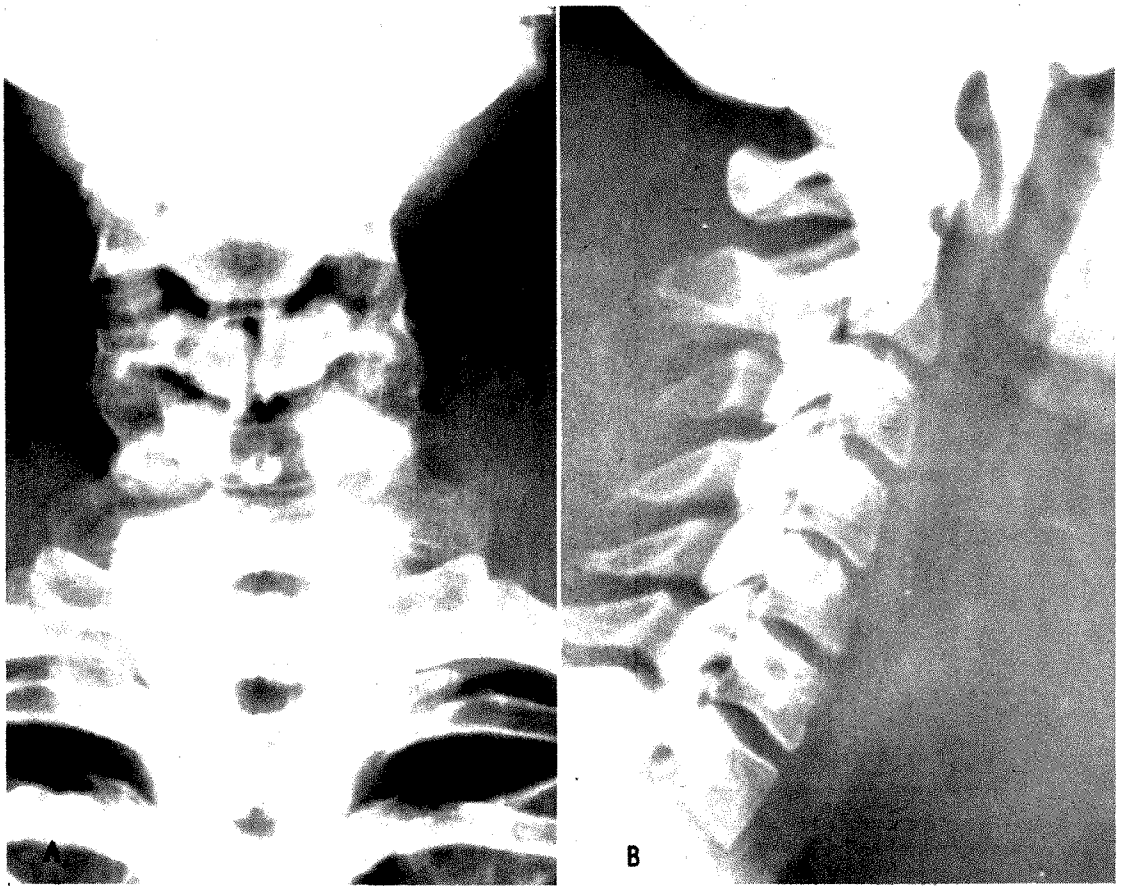

A
B

Fig. 2. (*A* and *B*) Roentgenograms taken one month after Figure 1.

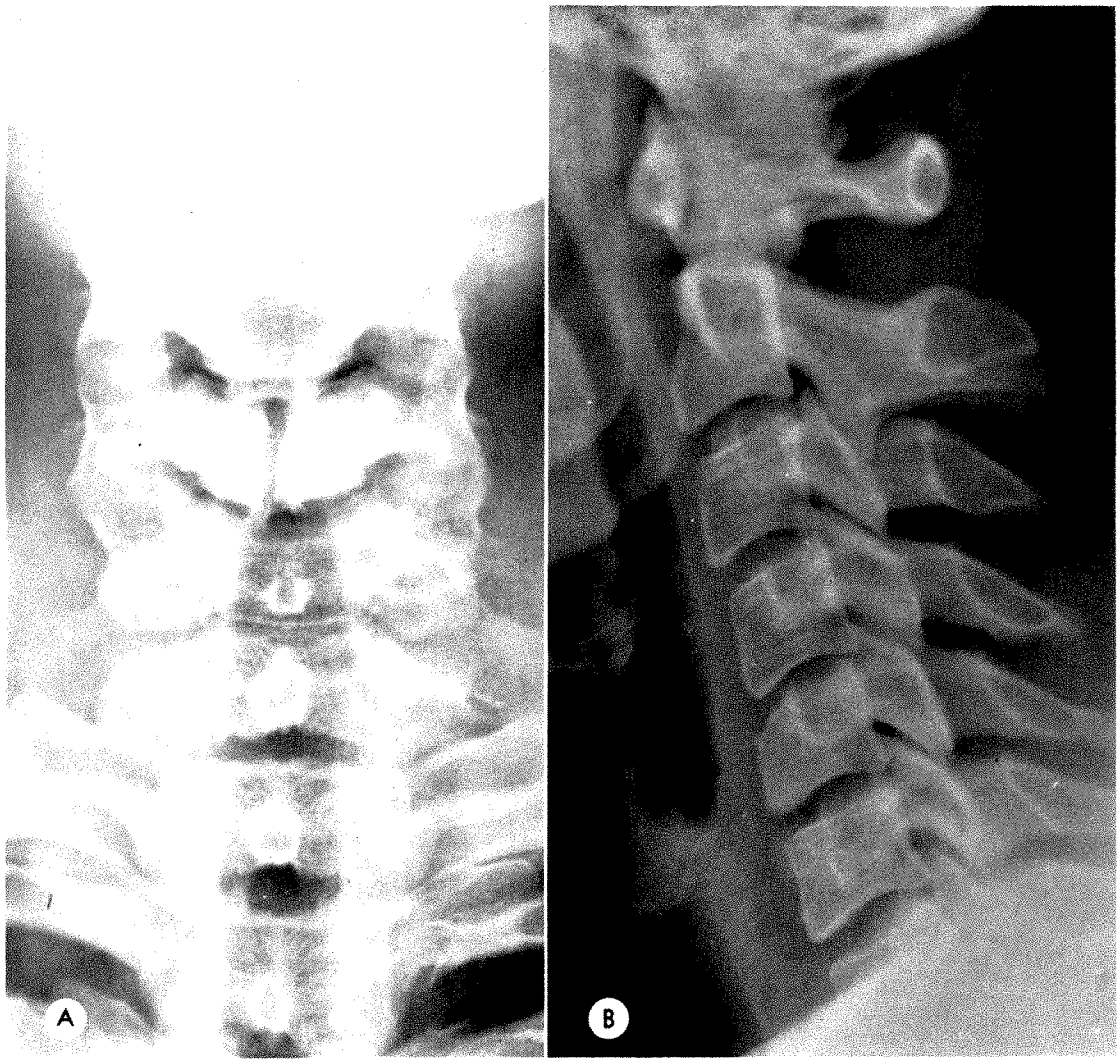


Fig. 4. Roentgenograms taken almost three months after Figure 1. (*A*) There is further decrease in the anterior height of C5. (*B*) A slight amount of bony bridging is present.

Fig. 3. (*A* and *B*) Roentgenograms taken approximately two months after Figure 1.

The patient was placed in cervical halter traction, which was followed later by use of a cervical brace. Periodic roentgenograms were taken (Fig. 2, *A* and *B*; and 3, *A* and *B*).

When he was last seen eighty-five days after admission, he still had no neurologic symptoms. The roentgenograms made at that time showed further decrease in the anterior height of C_5 (Fig. 4*A*). Some bony bridging of the fracture fragments was also present (Fig. 4*B*).

COMMENT

The patient does not remember receiving a blow to the occipital region. However, there had been a large amount of luggage stacked in the back of his small car and one of these pieces may have become a missile. The patient's neck probably went through the same motions as are carried out in a "whiplash" injury. It would be of interest to know what part the blow to the occipital region contributed to the end result.

The possibilities of the radiolucent line in the vertebra being air in the trachea, a failure of union of the vertebral body or a spina bifida occulta were considered. The first was ruled out because of the roentgen appearance and the downward displacement of the left half of the vertebral body. The last two were eliminated on the basis of a routine army induction chest roentgenogram which had been taken approximately two years prior to the accident and on which C_5 could be fairly well visualized and appeared to be normal.

REVIEW

A review of American and English literature for the past twenty years failed to produce a documented case of a vertical fracture and without neurologic symptoms, although mention of such a case was made by Ellis.[1] The possible mechanisms of vertical fractures have been discussed thoroughly by Richman and Friedman.[2]

SUMMARY

A case of a vertical fracture of a cervical vertebra with no neurologic symptoms has been presented.

Department of Radiology
Offutt AFB Hospital
Omaha 13, Nebraska

The author wishes to thank Doctors S. P. Peartree, W. W. Jurgensen and R. McDonald of Omaha, Nebraska, for their consultation.

REFERENCES

1. Ellis, V. H. Injuries of cervical vertebrae; President's address. *Proc. Roy. Soc. Med.*, 1946, *40*, 19–26.
2. Richman, S., and Friedman, R. L. Vertical fracture of cervical vertebral bodies. *Radiology*, 1954, *62*, 536–542.

VOL. 86, NO. 2

LAMINAGRAPHY OF THE TEMPORAL BONE*

By S. BRÜNNER, ØJVIND PETERSEN, *and* P. STOKSTED
GENTOFTE, DENMARK

ROENTGEN examination is a valuable aid in diagnosing diseases of the ear associated with changes in the osseous structure. The roentgenograms must be taken with the patient in positions designed to reveal abnormalities in the area most frequently involved in chronic ear disease, *viz.*, the attic-aditus-antrum area. In projections such as Schüller's, Stenvers', Runström's, Chaussé's, etc. it is possible to demonstrate major or minor parts of the inner aspect of the temporal bone. Better results are sometimes obtained by supplementing the examination with stereoscopic views.

With plain roentgenography of the temporal bone, it is impossible to eliminate visualization of the surrounding structures which will blur the delicate details in the anatomy of the inner ear. The only way of visualizing the inner aspect of the temporal bone without also reproducing the interfering structures on the film is by laminagraphy.

Numerous laminagraphic studies of the temporal bone have been reported since Zeidses des Plantes[10] and Bartelink[1] employed a curvilinear laminagraph in a few cases. Most investigators, however, have used linear laminagraphy, although, theoretically, laminagraphy with polycyclic movement gives the most exact laminagraphic plane with blurring of the surroundings, in contradistinction to rectilinear laminagraphy, which only blurs perpendicular to the tube movements.

EQUIPMENT

In 1950, Sans and Porcher[7] designed a laminagraph called the Polytome, which makes it possible to use four types of obscuring movements. The trajectories are linear, circular, elliptic, and "hypocycloidale." Table 1 shows the various specifications of the Polytome.

The roentgen-ray table may be tilted into any position between horizontal and vertical. The distance between the focus and the plane of cut is constant, 110 cm. The distance from the film to the plane of the cut is, however, variable, since two cassette holders may be used, one placed at the minimum distance from the plane of cut (magnification 1.3) and the other at the maximum (magnification 1.6). The tube is furnished with a fine focus (0.3×0.3 mm.) and a 1×1 mm. focus. The cassette holder at the minimum distance from the plane of cut is provided with a so-called "rotating grid." The cassette holder with the magnification of 1.6 can be inclined.

Owing to these advantages, we chose the Polytome for laminagraphy of the temporal bone, selecting the polycyclic trajectory, named "hypocycloidale" by the manufacturers.

TECHNIQUE AND FINDINGS

Various projections have been used for laminagraphy of the temporal bone. James[4] has described an anteroposterior and an oblique lateral view; Mündnich and Frey[6] reported no less than 5 different projections, *viz.*, an anteroposterior, a lateral, a longitudinal and an axial view of the pyramid, and an axial view of the head. According to our experience with about 250 laminagraphies of the ear, the anteroposterior and lateral projections of the temporal bone are the most useful. In our opinion, these two views give, in most cases, sufficient detail of the temporal bone. Another reason for restricting laminagraphy to one or two projections is to reduce the roentgen-ray dose, as each projection requires six to ten cuts.

* From the Department of Radiology and Department of Otology, Copenhagen County Hospital, Gentofte, Denmark.
Aided by a grant from the King Christian X Foundation.

TABLE I

SPECIFICATIONS OF THE POLYTOME*

Angle of Cut	Linear† Trajectory	CD	Elliptic Trajectory	CD	Circular Trajectory	CD	Hypocycloidal Trajectory	CD	SC
29°	56 cm.	1			176 cm.	3.1			2.3 mm.
36°	69 cm.	1			217 cm.	3.1			1.8 mm.
40°	77 cm.	1	186 cm.	2.4					1.6 mm.
48°	92 cm.	1					451 cm.	4.9	1.3 mm.
60°	115 cm.	1							1.0 mm.

* The table lists, for each angle of cut, the various trajectories, the length of each at the level of the roentgen-ray tube focus, and the minimum coefficient of distinctness (CD) of the image obtained, determined by the relation of each complex trajectory to the linear trajectory of the same angular opening. SC means separating capacity (thickness of cut). It varies with the angle of cut. The exposure time is proportional to the trajectory.

† In linear trajectory, all angles of cut from 10° to 60° can be obtained.

ANTEROPOSTERIOR PROJECTION

With the patient supine and the median plane of the head vertical to the table, a line from the lateral angle of the eye to the tragus also should be vertical to the roentgen-ray table (Fig. 1). The area to be visualized extends approximately from the root of the tragus 15 mm. to the posterior aspect.

Anteroposterior laminagraphy of the normal temporal bone (Fig. 2–6) will reveal in the most ventral cut, taken on a level with the tragus, the external auditory meatus (1) as the most characteristic structure. This may be used as an anatomic point of reference for the initial, cursory study of the laminagram. It is delimited inferiorly by a solid bony shadow representing the tympanic part of the temporal bone, and superiorly by the upper posterior area of the osseous part of the auditory meatus, the so-called "spur" (2) with the tympanic groove. The spur ends anteriorly in the attic as a prominent, broad-based wedge. In cases of lesions in the attic, the spur assumes a rounded shape or an irregular, eroded surface. Thus, this detail of the laminagram should always be inspected very closely.

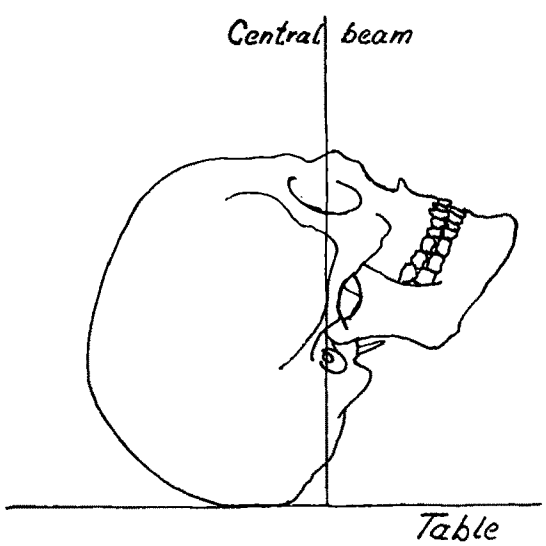


FIG. 1. Diagram showing the direction of the central beam in anteroposterior laminagraphy. (Reproduced with permission from the *Archives of Otolaryngology*.)

The medial part of the external auditory meatus (1) is delimited by the tympanic membrane, normally manifesting itself as a faintly streaked contour. It continues superiorly in the handle of the malleus. The head of the malleus (m) rises in the middle of the attic as a club-shaped shadow. The tympanic cavity (3) presents on the most ventral laminagraphic cuts as a sharply outlined profile, affording good visualization of the hypotympanic recess and promontory, with the spiral-shaped cochlea (4) shown medially. At the site of the upper part of the promontory, just above the cochlea, a portion of the facial

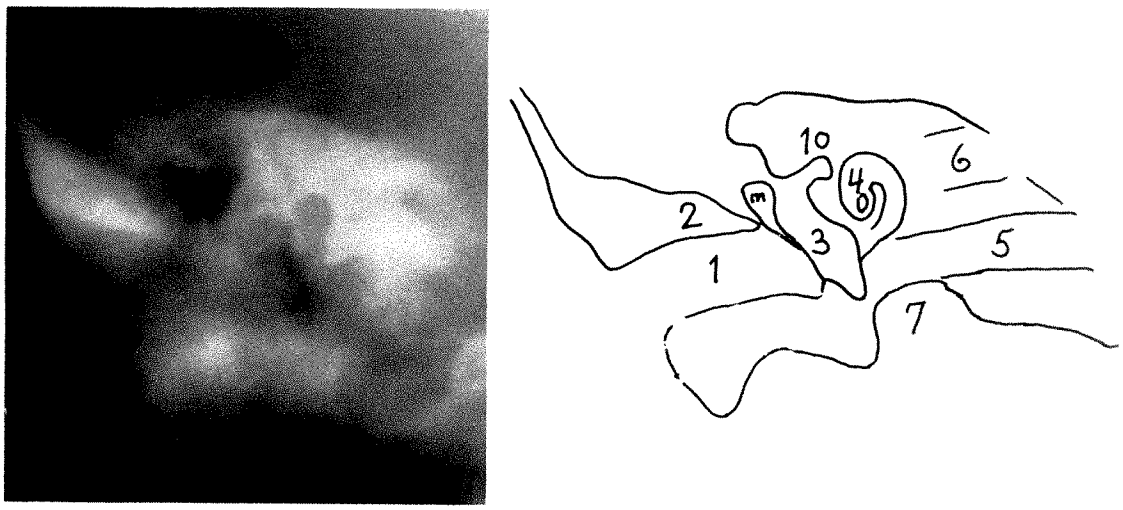


Fig. 2. Anteroposterior laminagram and diagram of a cut through the anterior part of the tympanic cavity (3), showing the malleus (m), spur (2), cochlea (4), external (1) and internal (6) auditory meatuses, pharyngo-tympanic tube (5), jugular fossa (7) and part of the canal for the facial nerve (10). (Reproduced with permission from the *Archives of Otolaryngology*.)

canal (10) is visible, *viz.*, its course from the opening for the facial nerve at the bottom of the internal auditory meatus (6) to the promontory.

The tympanic cavity continues in the attic sharply outlined laterally against the spur and showing on its medial aspect the prominence of the canal for the facial nerve and the lateral semicircular canal (8). Superiorly, the roof is made up of the tegmen tympani, and, since this is only faintly visible or entirely absent on the laminagrams, this lack of visualization need not be a sign of a pathologic condition.

Medial to the cochlea is the internal auditory meatus (6) which appears broad and distinct on the most anterior cuts. In the lateral part of the internal auditory meatus, a small bony crest, the transverse crest, is sometimes visible. Below the in-

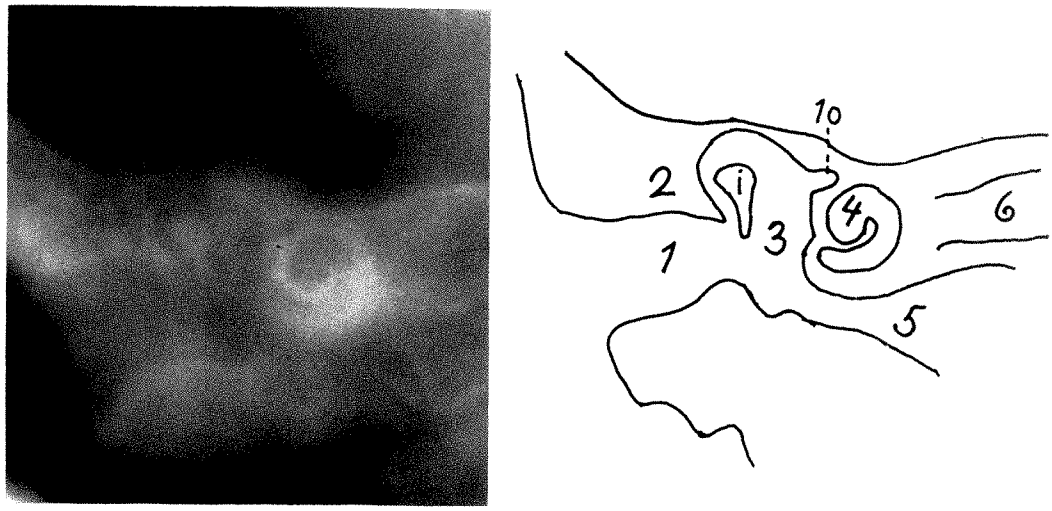


Fig. 3. Anteroposterior laminagram and diagram of a cut made more posteriorly in the tympanic cavity (3) than Figure 2 in which, in addition to the structures shown in Figure 2, the incus (i) may be seen. (Reproduced with permission from the *Archives of Otolaryngology*.)



Fig. 4. Anteroposterior laminagram and diagram of a cut through the vertical part of the canal for the facial nerve (11), also showing the styloid process (12). (Reproduced with permission from the *Archives of Otolaryngology*.)

ternal auditory meatus, the pharyngotympanic tube (5) extends from the floor of the tympanic cavity.

On the inferior surface of the pyramid, a single structure may be seen just beneath and medial to the hypotympanic recess, *viz.*, the jugular fossa (7) accommodating the bulb of the jugular vein.

On sections a few millimeters posterior to the tragus, all these structures are generally visible, but the tympanic membrane and malleus (m) have disappeared. Instead, the sections visualize the incus (i) shaped as a short club. Unlike the malleus, the incus is in an extreme lateral position in the attic, its lateral contour extending parallel to the spur. The situation of the two ossicles is extremely important, partly for identification purposes and partly for diagnosing dislocations and congenital deformities. Somewhat more posteriorly, the vertical course of the facial canal (11) may

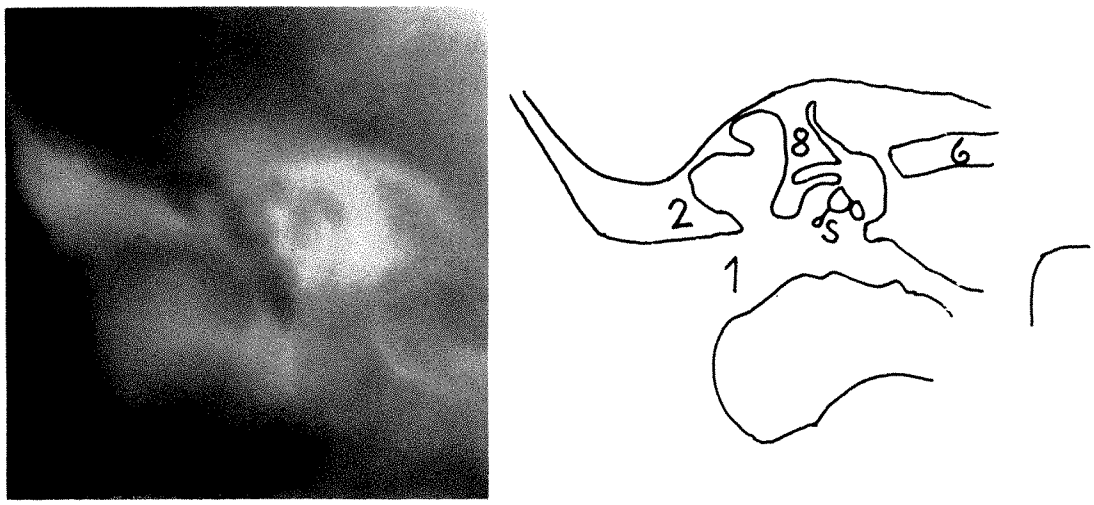


Fig. 5. Anteroposterior laminagram and diagram of a section in the posterior part of the tympanic cavity showing the stapes (s), vestibule, and semicircular canals (8). (Reproduced with permission from the *Archives of Otolaryngology*.)

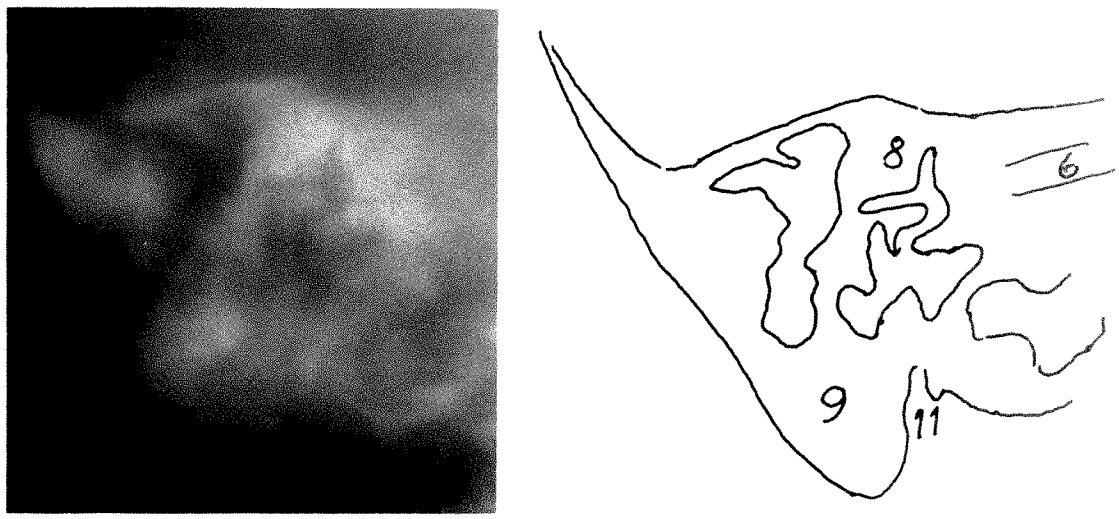


Fig. 6. Anteroposterior laminagram and diagram of a cut through the mastoid antrum (9) and semicircular canals (8). (Reproduced with permission from the *Archives of Otolaryngology*.)

be seen extending down from the bottom of the tympanic cavity, with its outlet on the inferior surface of the pyramid in the stylomastoid foramen (12).

Cuts just behind the cochlea show the vestibule and semicircular canals (8) most distinctly. The same cut shows the smallest "long bone" of the body, the stapes (s), in part or in full, with the footplate in the oval window which is delimited inferiorly by the subiculum, which, in turn, forms the upper limit of the round window. As yet, we have not found changes indicating ankylosis of the stapes.

On these last-mentioned cuts there is still visible a distinct outline of the attic, or rather the aditus of the antrum, whereas the external as well as internal auditory meatuses have decreased both in size and sharpness, because their posterior bony limit is being reached.

Cuts on a level with the mastoid process (9) still show part of the vestibule and semicircular canals and, at times, a small portion of the internal auditory meatus, although now the external auditory meatus has completely disappeared from view, being replaced by the pneumatic cells of the mastoid process and, superiorly, by the mastoid antrum.

LATERAL PROJECTION

In this projection, the patient is prone with the head turned so that the ear to be examined is up and with both external auditory meatuses in a line perpendicular to the table (Fig. 7). In order to make the patient more comfortable, the shoulder on the side to be examined is elevated, with the homolateral arm flexed and the contralateral arm extended.

Cuts are obtained from the plane of the tympanic membrane, *viz.*, about 2 to 2.5 cm. below the level of the mastoid process and 1 cm. medial, as a rule at a distance of 1 mm. The first cuts visualize the ossi-

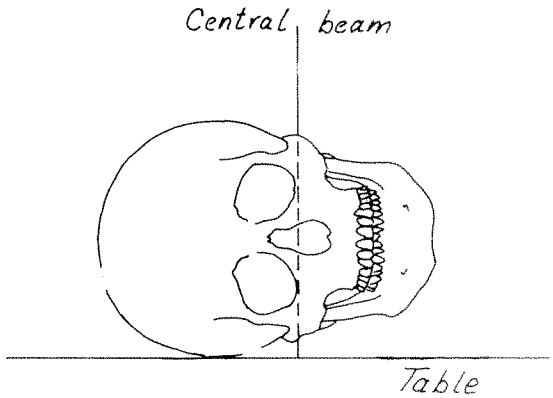


Fig. 7. Diagram showing the direction of the central beam in lateral laminagraphy.

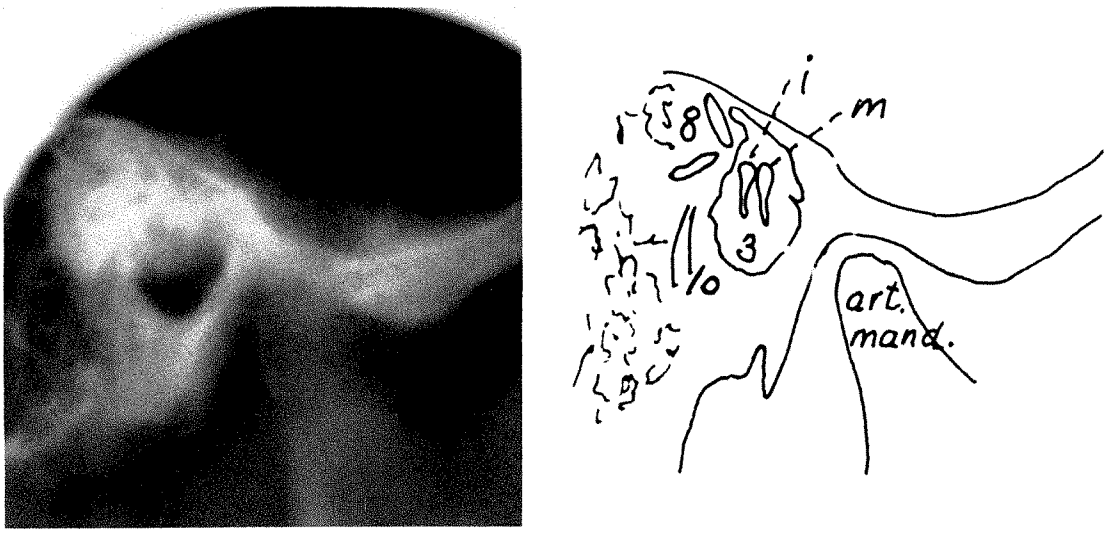


Fig. 8. Lateral laminagram and diagram of a cut through the external part of the tympanic cavity (3), showing the malleus (m) and incus (i) as well as part of the semicircular canal (8) and facial canal (10).

cles (Fig. 8 and 9), the bodies of which are in the attic. The malleus (m) is the most anterior one, its handle projecting down into the tympanic cavity. The body of the incus (i) is immediately posterior to the head of the malleus. The handle of the malleus and the long process of the incus are parallel. Sometimes, the last link of the chain, the stapes may be discerned against the oval window. Superiorly to this, part of the semicircular canal (8) is visualized. Between the latter structure and the tympanic cavity, the upper one-third of the facial canal (10) is often visible. Cuts made more medially will show the distal part of this canal (12).

ILLUSTRATIVE CASES

To demonstrate the diagnostic value of the roentgenographic techniques described

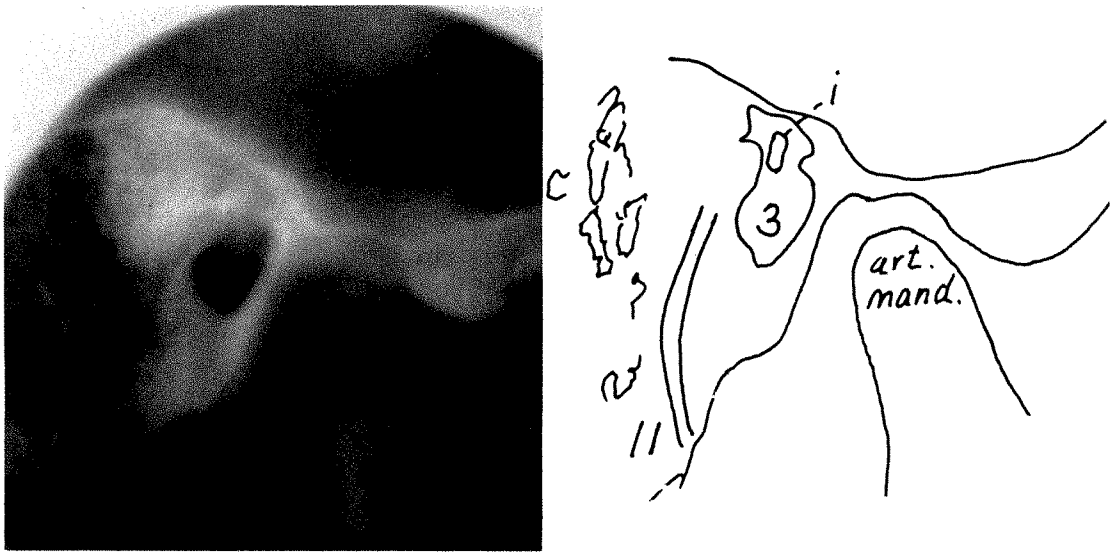


Fig. 9. Lateral laminagram and diagram of a cut through the medial part of the tympanic cavity (3) visualizing the vertical part of the facial canal with the styloid foramen (11).

above, 2 characteristic cases are reported in which laminagraphy was of primary diagnostic importance.

Case 1. This woman, aged seventy years, had been suffering since childhood from chronic, bilateral otitis media with periodic suppuration from both sides when she had colds and sinusitis. Her hearing had been evenly deteriorating, so that for some years she had been using a hearing aid on the left. For the past five years she had had episodes of gyratory and nautic vertigo as well as left-sided tinnitus.

About five months prior to admission, she had experienced a period of severe fatigue, during which total left-sided facial palsy developed within the course of a few days. The condition was interpreted as the sequel to cerebral hemorrhage, and the patient was treated only by bed rest at home. She was admitted for cholelithiasis but, because of the facial palsy, the following investigations were performed.

Neurologic Examination. Results showed left-sided peripheral facial palsy and no sense of taste on the anterior two-thirds of the tongue, the left pupil larger than the right and inactive to light, and diffuse weakening of the limbs with generalized weakening of all reflexes. Romberg's test showed a tendency to fall backward. Cerebrospinal fluid examination was normal. It was difficult to interpret the findings, but an arteriosclerotic lesion in the brain stem was believed probably to be responsible.

Ophthalmologic Examination. This revealed sclerotic changes of the fundus and left-sided hypesthesia of the cornea, presumably due to lagophthalmos. The previously diagnosed ophthalmoplegia interna could not be re-demonstrated, so it must have been transient or drug-induced.

Otologic Examination. There were large, apparently central defects of both tympanic membranes but no discharge or sign of cholesteatoma. The nasolacrimal reflex was present and equal on both sides. An audiogram showed bilateral hearing loss of the conduction type, and nerve deafness consistent with her age. The stapedial reflex could not be tested because of the bilateral chronic otitis.

Roentgen Examination. Using Schüller's, Towne's (slit exposure), and Stenvers' projections, reduced pneumatization with sclerosis of the cells on both sides was revealed but there were no signs of cavitation. Laminagraphy disclosed cavitation on the left side at the site of the anterior portion of the tympanic cavity and the attic, with destruction of the superolateral corner of the cochlea in the horizontal course of the facial canal as well as dilatation of the lateral portion of the internal auditory meatus (Fig. 10 and 11).

Operative Findings. On the basis of the roentgen findings, the patient was subjected to resection of the mastoid process and tympanic cavity on the left with decompression of the left facial nerve. There was pronounced osteitis of the incus, both superficial and profound, and a cholesteatoma in the anterior part of the antrum extending down into the upper portion of the tympanic cavity. At the side of the spur, the facial canal was surrounded by osteitic bone with ample granulation tissue on the sheath.

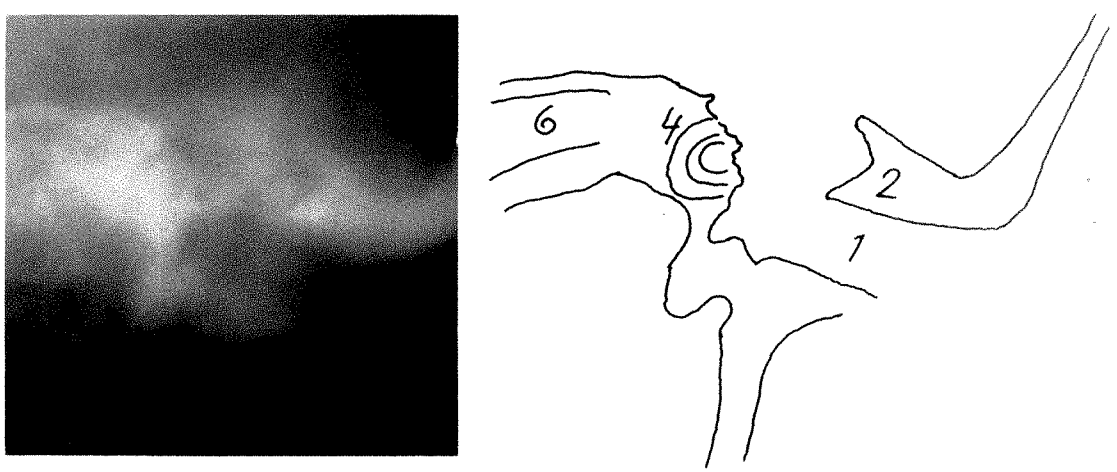


FIG. 10. Case 1. Anteroposterior laminagram and diagram showing destruction at the site of the second part of the facial canal; external auditory meatus (1), spur (2), cochlea (4), internal auditory meatus (6).

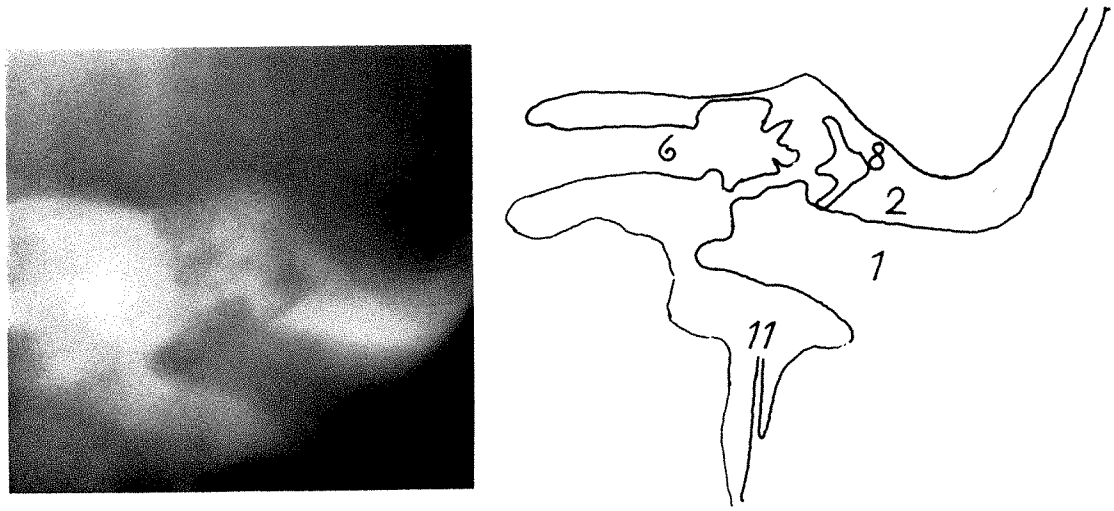


Fig. 11. Case 1. Anteroposterior laminagram and diagram, made more posteriorly than Figure 10, showing that the destruction extends into the lateral part of the internal auditory meatus in the area of the facial nerve; external auditory meatus (1), spur (2), internal auditory meatus (6), semicircular canal (8), facial canal with the styloid foramen (11).

The nerve was hyperemic and showed violent swelling when exposed. It was exposed upwards as far as the geniculate ganglion and downwards as far as the stylomastoid foramen, but no abnormalities were found in these sites.

Comment. In recent years, developments in the field of otology, including the increased number of operations performed to improve hearing and the correlative increase in interventions on the facial canal, are making ever greater demands on diagnostic roentgenology.

Routine studies of the temporal bone in the various projections (such as Schüller's, Chaussé's, Stenvers', Runström's, and Towne's—as well as Law's projection based upon those of Stenvers and Schüller) illustrate the pneumatization and disclose the presence of acute or chronic inflammatory changes. In the diagnosis of cholesteatoma or of changes in the ossicles or the facial canal, ordinary roentgen investigation often fails. In 1936, MacMillan[5] reported destructive changes of the temporal bone in only 45 per cent of 288 confirmed cholesteatomas when studying the patients in Law's projection, and in 60 per cent when using Towne's projection. Chiat and Kittredge[3] found destructive changes in 50 per cent also in Towne's projection. In 1960, Brünner, Jensen and Jespersen,[2] in 80 verified cases of cholesteatoma, correctly diagnosed only 46 per cent using Schüller's and the Runström III projections. By employing Chaussé's III projection, the correct diagnoses could have been increased to 70 per cent since this projection increases the possibility of visualizing the antrum and attic, distinct from the labyrinth, and also shows the lateral wall of the attic and external auditory meatus.

By supplementing the usual procedures with laminagraphy, the possibility of demonstrating cholesteatoma could be increased to 80 per cent. This conclusion is supported by a report of Tarp,[8] who demonstrated cholesteatoma by laminagraphy in 19 of 22 verified cases. Thus, laminagraphic study is of great importance, as it affords an opportunity for further advances, especially in visualizing the antrum and/or attic as well as the superior aspect of the external auditory wall and the lateral wall of the attic. In other words, laminagraphy permits a much more exact location of the destruction as well as the damage wrought by it.

There are three important, characteristic findings in the laminagraphic demonstration of changes caused by cholesteatoma: (1) enlargement of the attic and/or antrum and simultaneous destructive changes of the medial or lateral wall of the attic. It has been emphasized by Tarp[8] that enlargement of the antrum to more than 10×12 mm. should arouse suspicion of a cholesteatoma cavity. In our experience, the extent of this enlargement is often very difficult to assess. In order to facilitate the evaluation we introduced the practice, one year ago, of examining both temporal bones by laminagraphy in the anteroposterior projection on the same film. This has supplied us with an extremely important diagnostic aid in evaluating the size of the attic and/or antrum; (2) destruction of the upper medial portion of the external auditory meatus (the spur); in most cases of cholesteatoma involving the attic, it undergoes destruction, while in the presence of changes in the more posterior part of the antral region the spur is often intact; all cases of destroyed spur ought to arouse a suspicion of cholesteatoma and/or osteitis; and (3) destructive change or fragmentation of the ossicles, due in most cases to compromised blood supply, especially to the incus.

Laminagraphy usually visualizes the facial nerve in its entire course through the temporal bone, thus being of valuable supplementary aid in the topical diagnosis of facial nerve lesions. In Case 1, there were destructive changes of the medial wall of the attic as well as around or above the second segment of the facial nerve.

As already mentioned, the projections in current use (Stenvers', Runström's, and Towne's) did not reveal changes other than reduced pneumatization and sclerosis of the cellular systems on both sides, but with no signs of cavitation. Laminagraphy was required to demonstrate the presence of destructive cavitation, especially at the site corresponding to the course of the facial nerve. Surgery could be instituted after the lesion had been fairly accurately located.

CASE II. This twenty-three year old man, formerly a sailor, had had a bilateral mastoidectomy at the age of two. Since then, his hearing had been impaired, but he had never had aural discharge, attacks of fever, pain, or recurrent otitis.

Clinical Examination. His general condition

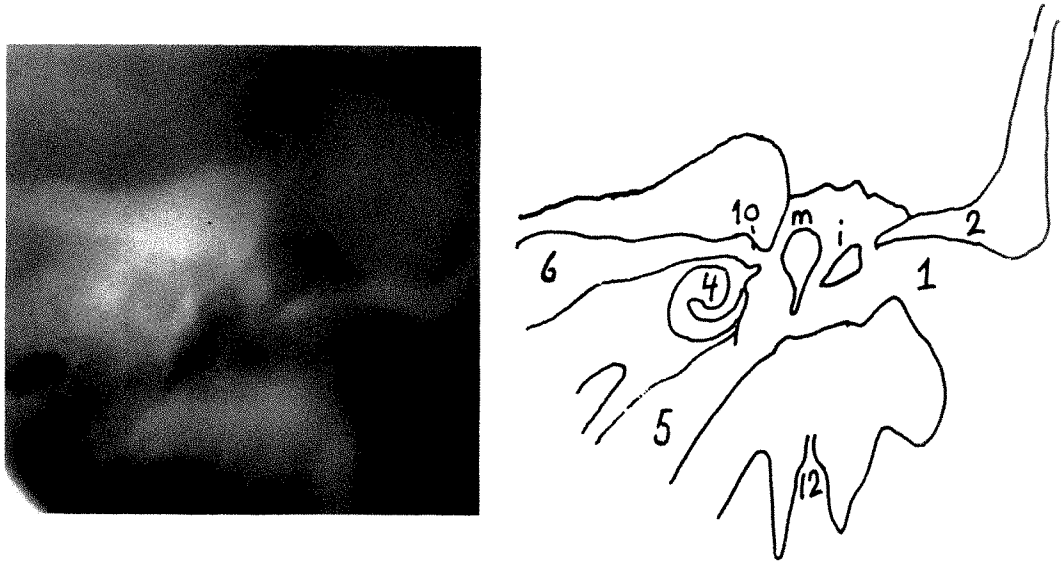


FIG. 12. Case II. Anteroposterior laminagram and diagram of a cut through the anterior part of the tympanic cavity; external auditory meatus (1), spur (2), incus (i), malleus (m), cochlea (4), pharyngotympanic tube (5), internal auditory meatus (6), jugular fossa (7), facial canal (10), styloid process (12).

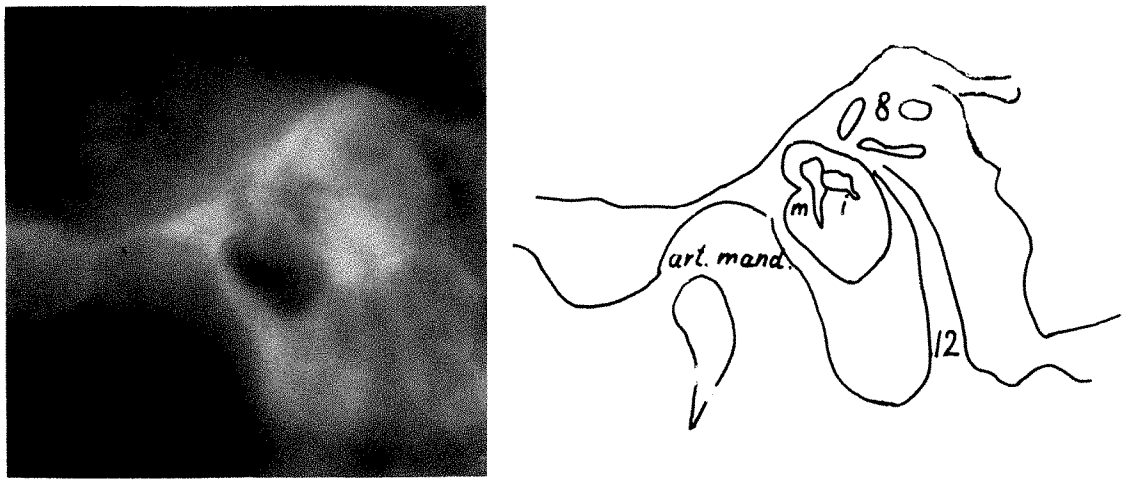


Fig. 13. Case II. Lateral laminagram and diagram of a cut through the tympanic cavity, showing the dislocation of the incus (i); malleus (m), semicircular canal (8), styloid process (12).

was good, although mentally he was severely disturbed by this handicap, and his speech was indistinct and monotonous.

Otologic Examination. The tympanic membranes were grey and lustreless, but intact; no discharge was present in the auditory meatuses. There was hearing of the whispered voice at 0.1 m. and of the spoken voice at 0.5 m. in both ears.

Roentgen Examination. The temporal bones, in the standard projections used in Case 1, showed the sequelae of mastoidectomy on both sides with no signs of destructive changes. Anteroposterior and lateral laminagraphy (Fig. 12 and 13) showed on the left that the body of the incus had slid down, so that the long process of the incus was horizontal. It was impossible to locate the stapes definitely. On the right side, the head of the malleus was distinct, but the body of the incus and its long process were blurred and appeared to be dislocated. On both sides operative cavities left by simple mastoidectomy were seen.

Operative Findings. Surgery was performed on the left ear, on October 17, 1959. After exposing the middle ear, the incus was seen to be displaced, with its long process pointing outward and forward instead of downward and medially. This interrupted the connection between the incus and stapes, there being a distance of 2–3 mm. between the two joint surfaces. It proved possible to reduce the incus and successfully restore the normal communication between the joints.

The postoperative course was uneventful despite the fact that, while incising the auditory meatus, an opening into the antrum could not be avoided, because the posterior wall of the external auditory meatus had been removed at the previous operation. The patient's hearing was restored to practically normal, with the hearing of the whispered voice increasing from 0.10 m. to 4.5 m.

Operation on the right ear was performed on November 13, 1959. After opening the middle ear, it was found that the situation was less favorable than it had been on the left, the body of the incus being turned posteriorly almost 90°. This had completely interrupted the connection between the malleus and stapes, and the incus was firmly impacted between the facial canal and the lateral wall of the attic. Restoring the ossicle chain was out of the question. Instead, the tympanic ring was freed from the tympanic groove in the posterior two-thirds of the circumference, so that the tympanic membrane could be wrapped around the intact and freely mobile stapes.

The hearing of the whispered voice improved from 0.1 m. to 2 m. within three weeks. In other words, the hearing improvement in this ear too was of great practical importance.

Comment. Today it is of the utmost importance to be able to diagnose morbid processes in the temporal bone at a stage so early that inoperable changes of the facial nerve and vestibular apparatus may be prevented. If plastic operations for improving the hearing are to give a favorable

result, the processes must not have had time to interrupt the blood supply to the ossicles or to cause major bone destruction, since it is the object of tympanoplastic operations to reconstruct an intact chain of ossicles and restore to the patient a degree of hearing sufficient for practical purposes.

The increase in recent years in the frequency of cranial injuries due to traffic accidents resulting in fractures of the base of the skull has also increased the number of traumatic changes in the ossicles and their conduction links. As early as 1955, Welin[9] stressed the great importance of laminagraphy of the ossicles in cases of infection and especially in traumatic injuries. It is beyond doubt that, in numerous cases of cranial injury, more attention has been devoted to the immediate vital problems of a more general nature at the cost of the otologic changes which have led to hearing impairment.

Hearing impairment following operations performed a number of years ago for otitis was probably due in many cases to the less advanced operative and anesthetic techniques of those times. This is illustrated by our Case II. In childhood the patient had undergone simple bilateral mastoidectomy after which his hearing had been greatly impaired. Laminagraphic investigation revealed displacement of the ossicles, with dislocation in the incostapedial joint. Bilateral, exploratory tympanectomy revealed, in accordance with the roentgen findings, dislocation of the incus on both sides. After reduction of the left incus, an anatomically correct position of the ossicles was obtained. On the right, the incus could not be reduced, as it was firmly impacted. Instead, myringostapediopexy was performed. The operative treatment improved the hearing in the right ear considerably, but also to some extent in the right ear, so that almost normal hearing was restored.

Laminagraphy of the ossicles will no doubt gain ever increasing practical importance, not least because of the increased number of traffic accidents. It will contribute to diagnosing displacement of the ossicles caused by the more primitive techniques formerly used in aural surgery.

SUMMARY

A laminagraph with polycyclic movements and excursions wide enough to permit thin and well-defined cuts is discussed. By means of this apparatus, anteroposterior as well as lateral views of the temporal bones were obtained. A description of the normal sections and the characteristic pathologic findings is given.

In connection with a case of cholesteatoma, the most characteristic findings are stressed: (1) destruction of the spur; (2) dilatation of the antrum and/or attic demonstrated by comparison with the normal side; and (3) destruction or fragmentation of the ossicles.

A case of dislocation of the ossicles illustrates the importance of lateral laminagraphy in diagnosing changes in the position of the ossicles.

Øjvind Petersen, M.D.
Copenhagen County Hospital
Gentofte, Denmark

REFERENCES

1. BARTELINK, D. L. Röntgenschnitte. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1933, *47*, 399–407.
2. BRÜNNER, S., JENSEN, G., and JESPERSEN, C. Value of different projections in diagnosing cholesteatoma. *Acta radiol.*, 1960, *54*, 177–185.
3. CHIAT, H., and KITTREDGE, R. D. Cholesteatoma of temporal bone. *Radiology*, 1958, *71*, 559–562.
4. JAMES, D. C. Tomography of temporal bone in disease of middle ear. *Brit. J. Radiol.*, 1957, *30*, 148–152.
5. MACMILLAN, A. S. Cholesteatoma in chronic otitis media. AM. J. ROENTGENOL. & RAD. THERAPY, 1936, *36*, 747–750.
6. MÜNDNICH and FREY. The Tomogram of the Ear. Georg Thieme, Stuttgart, 1959.
7. SANS and PORCHER. Polytome. *J. de radiol. et d'électrol.*, 1950, *31*, 300–302.
8. TARP, O. Tomography of temporal bone with polytome. *Acta radiol.*, 1959, *51*, 105–116.
9. WELIN, S. Radiology of temporal bone. *J. Laryng. & Otol.*, 1955, *69*, 515–525.
10. ZIEDSES DES PLANTES, B. G. Eine neue Methode zur Differenzierung in der Röntgenographie (Planigraphie). *Acta radiol.*, 1932, *13*, 182–192.

CYSTS AND TUMORS OF THE MANDIBLE*

THEIR DIFFERENTIAL DIAGNOSIS

By E. HOWARD JAYNE, M.D.,† ROBERT A. HAYS, M.D.,‡
and FREDERICK W. O'BRIEN, Jr., M.D.
CLEVELAND, OHIO

RADIOLOGISTS have over the years taken an increasing interest in roentgenography of the mandible, devoting their attention to systemic diseases and malignant tumors. While dentists and dental surgeons are primarily interested in odontogenesis, it is recognized that there is an overlapping of interests, and hence the roentgenographic appearances of odontogenic defects, mandibular diseases, and tumors should be equally familiar to both radiologists and their dental colleagues.

The mandible participates in three essential functions, *i.e.*, mastication, deglutition, and odontogenesis. Since odontogenesis is peculiar to the mandible, the pathologic changes occurring during this process must be recognized and carefully considered in the differential diagnosis.

Many classifications of mandibular cysts and tumors have been suggested. The difficulty in selecting an appropriate one stems from the complex histologic appearance of the lesions and the uncertainty of tissue of origin. In some tumors, such as ameloblastomas, there is no unanimity of opinion as to the benign or malignant potentiality of the entity. It is not the authors' intent to introduce another classification, but instead, Table 1 is to be used to provide a systematic approach to the lesions that are more frequently encountered.

In this paper the common cysts and tumors affecting the mandible are reviewed and those characteristics that may aid in differential diagnosis are discussed.

EMBRYOLOGY

In the sixth fetal week epithelial or ectodermal cells in the embryonic area of the mandible begin to multiply and extend downward into the adjacent mesoderm (Fig. 1, *1*). The mesoderm then responds by forming collections of cells called dental papillae which grow up into the descending ectodermal cells (Fig. 1, *2*). As the two meet, the ectodermal cells form a caplike cover over the dental papillae—the ectodermal cells will form the enamel while the mesodermal dental papilla will form the dentin and pulp of the tooth. Each embryonic tooth is cut off from any connection to the surface epithelium by a membrane of vascular connective tissue (periodontal membrane) which grows upward and completely encloses the dental germ (Fig. 1, *3*). The periodontal membrane eventually becomes a specialized periosteum, lining the tooth socket and connecting the tooth to the mandible. The mandible proper is formed from the paired Meckel's cartilages, which meet anteriorly in the midline, forming the mandibular symphysis. As the tooth bud matures the ectodermal cells divide into three layers. Those immediately adjacent to the mesodermal papilla are the enamel cells or ameloblasts. The outer layer forms the external enamel epithelium while the middle layer differentiates into a stellate network and secretes a fluid enamel pulp (Fig. 1, *4–6*). It is at this stage of differentiation that anomalies of development commonly occur.

DISCUSSION

I. MANDIBULAR CYSTS

Most mandibular cysts, except for the rather controversial type that has been

* From the Department of Radiology, The Cleveland Clinic Foundation and The Frank E. Bunts Educational Institute, Cleveland, Ohio.
† Formerly Fellow; present address: Los Angeles County General Hospital, Los Angeles, California.
‡ Formerly Member of the Staff; present address: St. Vincent Hospital, Worcester, Massachusetts

called "traumatic," are epithelium-lined. These are thought to increase in size because of the tension of the fluid or semisolid contents;[15] thus, they are usually globular due to the equal pressure in all directions. The cysts may be either single or multiple and may coalesce or remain separate. Some contain calcific material which will often give a clue as to the roentgenologic diagnosis. They consist of four types.

1. *Periodontal or Radicular Cysts.* Infections of the tooth pulp often spread through the pulp canal into the adjacent mandible,[15] forming a dental granuloma which appears roentgenographically as an oval radiolucent area at the tip of the pulp canal. This inflammatory response causes proliferation of epithelial remnants in the periodontal membrane, with subsequent formation of epithelium-lined cysts (Fig. 2). On roentgenograms, it is extremely difficult to differentiate between a granuloma and these radicular cysts even though the latter are supposedly more radiolucent with a well circumscribed, often slightly sclerotic, margin.[7] Stafne[15] does not believe that granulomas and radicular cysts of equal size can

TABLE I

CYSTS AND TUMORS THAT AFFECT THE MANDIBLE

I. Cysts
   1. Periodontal or Radicular
   2. Squamous
   3. Dentigerous
   4. Traumatic
II. Tumors
   A. Odontogenic Tumors
      1. Ameloblastoma or Adamantinoma
      2. Odontoma
      3. Cementoma
      4. Enameloma
      5. Myxoma
      6. Fibroma
   B. Nonodontogenic Tumors
      1. Osteoma
      2. Osteoid Osteoma
      3. Chondroma
      4. Neurofibroma
      5. Hemangioma
   C. Tumors of Indeterminate Origin
      1. Giant Cell
      2. Fibrous Dysplasia
      3. Reticuloendotheliosis
      4. Infantile Cortical Hyperostosis
   D. Malignant Tumors
      1. Primary Malignant Tumors
      2. Metastases
      3. Extension from Surrounding Tissues

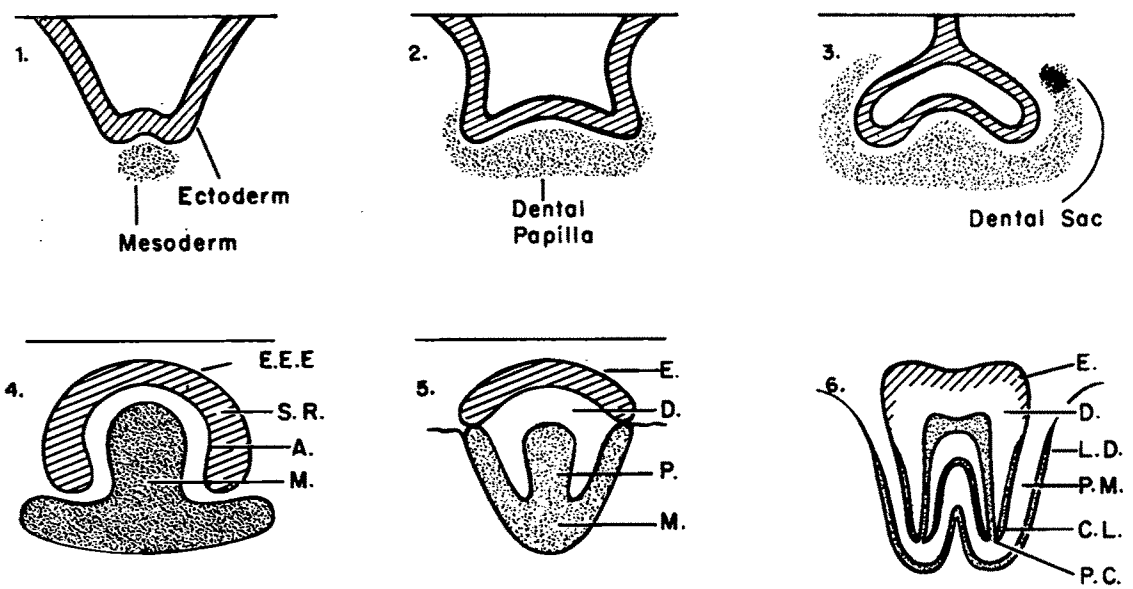


FIG. 1. Diagrams *1–6* illustrating embryologic development of a tooth. E.E.E.—external enamel epithelium; S.R.—stellate reticulum; A.—ameloblasta; M.—mesoderm; E.—enamel; D.—dentin; P.—pulp; L.D.—lamina dura; C.L.—cementoblastic layer; P.C.—pulp canal.



FIG. 2. A twenty-nine year old man had some aching in the right mandible. Following roentgenographic examination the cystic area was surgically opened and thick purulent material was expressed. The cavity was epithelium lined. The pathologic diagnosis was radicular cyst.

be differentiated by roentgen examination. The radicular cysts represent a common lesion, with the highest incidence in the third and fourth decades of life. More men than women are affected.

2. *Squamous Cysts.* In this category are placed the cysts that have no calcific deposits within them and are found at biopsy to have a squamous or epithelial lining. Any ectodermal tissue may give rise to squamous cysts. Thoma[17] suggests that these odontogenic cysts are formed from remnants of oral epithelium, proliferation of the dental lamina or from the enamel organ of a developing tooth. Jaffe[7] believes that these cysts are not associated with infection or maldevelopment of the dental germ but are formed when epithelial cells become trapped in the unfused mandible. The squamous cysts may be either monolocular or multilocular (Fig. 3 and 4). Usually, they have a relatively well-defined margin. In some cases the multilocular appearance is due to the lobulated margins of a single cavity (Fig. 5, *A* and *B*).

A rare type of squamous cyst, thought to have a different pathogenesis, is the primordial cyst that develops from the dental sac before any of the ectodermal or mesodermal cells become sufficiently differentiated to produce calcification. It is practically impossible to distinguish it from a common squamous cyst, being usually monocystic with a thin sclerotic margin. Primordial cysts are principally found in the second to sixth decades and affect both sexes equally. Thoma[17] believes that they are usually more common near the angle of the mandible where they sometimes extend into the ramus.

3. *Dentigerous Cysts.* Since these cysts develop from the enamel organ during or shortly after formation of the crown, they are associated with an unerupted tooth. Some authors[10,15] believe that these cysts are caused by an accumulation of fluid

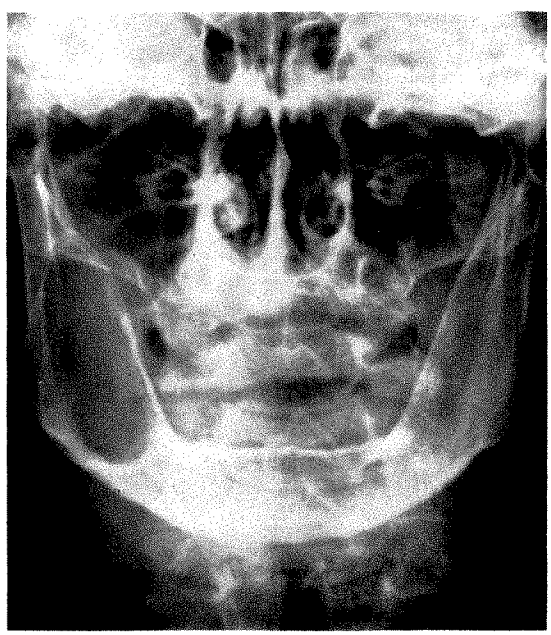


FIG. 3. A sixty-three year old woman with mandibular swelling for two years and local pain for one year. The pathologic diagnosis was squamous cyst. Note the unilocular appearance.

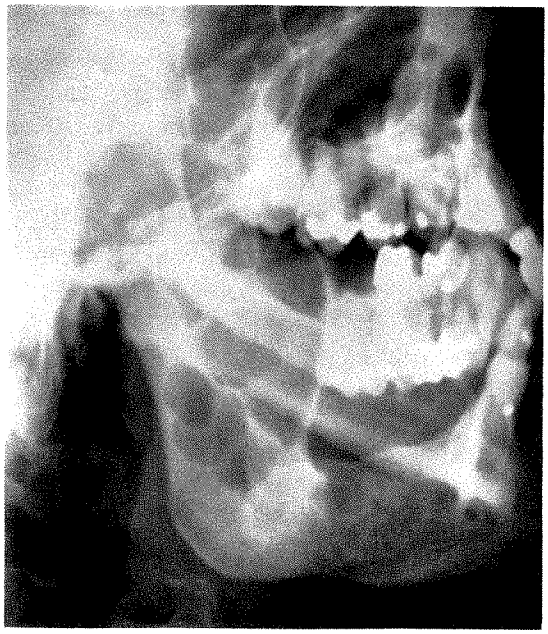


FIG. 4. A forty-four year old man with a two year history of aching in the left temporomandibular region when in a draft. The pathologic diagnosis was squamous cyst. Note the multilocular appearance.

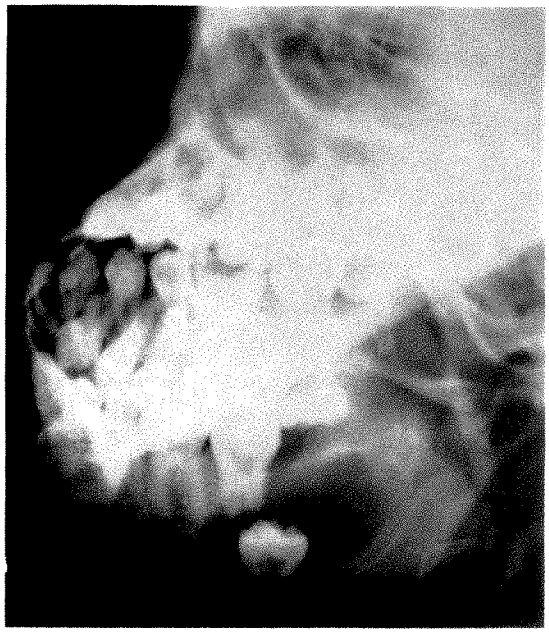


FIG. 6. An eighteen year old, asymptomatic female was found to have a cyst on routine dental roentgenograms. At operation the diagnosis of a dentigerous cyst was confirmed. Note the orientation of the cyst to the crown of the tooth.

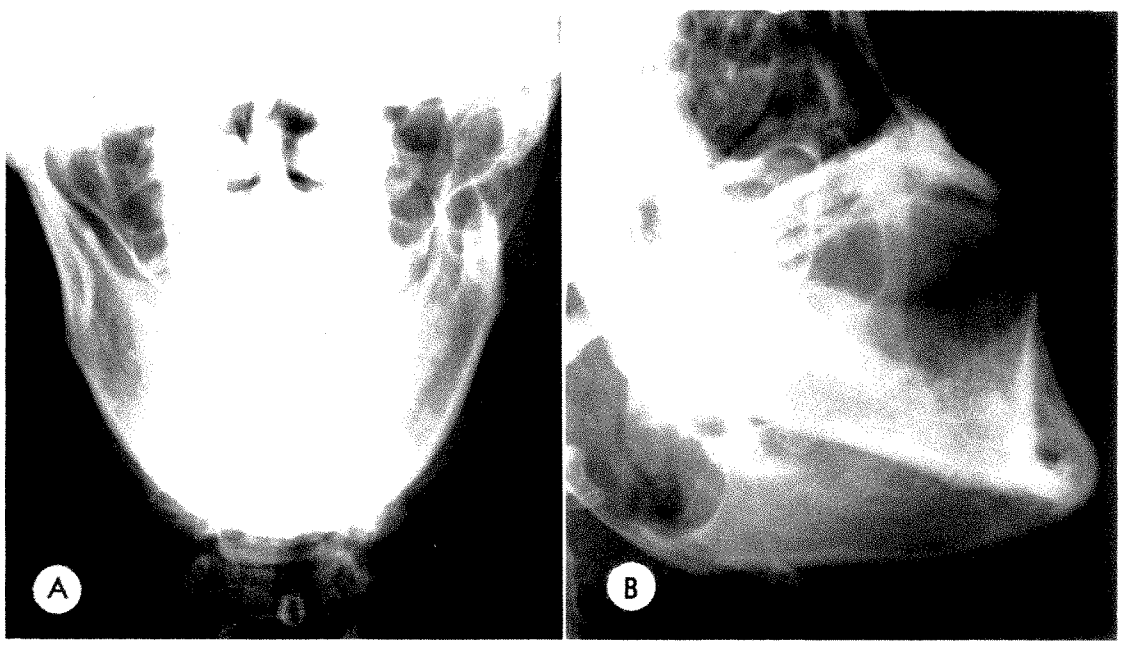


FIG. 5. A sixty-seven year old Negro woman with a fourteen year history of a left mandibular swelling. The pathologic diagnosis was squamous cyst. (*A*) Note the multilocular appearance on the postero-anterior roentgenogram. (*B*) Oblique roentgenogram showing the true single cavity.

between the enamel and the surrounding soft tissue capsule while others[7] think that the fluid within the enamel anlagen is responsible. In either event the onset is early in life, usually in the first two decades, and Stafne[15] is inclined to think that there is a hereditary tendency. The cysts are predominantly single. The roentgenographic appearance is that of a monolocular cystic lesion enveloping the crown of a normal appearing tooth (Fig. 6). Usually there is a clearly defined margin, and those in the molar region are more likely to have a sclerotic rim.[14]

4. *"Traumatic" or Nonepithelium-lined Cysts.* These cysts are lined with connective tissue instead of epithelium. One opinion[15] is that trauma may be the etiologic factor causing an intramedullary hematoma which as it degenerates leaves a cystic area. There is some doubt that trauma is always the etiology, and it has been suggested that these cysts may result from an aberration in growth and development of the local osseous tissue.[7] The borders of these cysts are not well defined and are more irregular than those of epithelium-lined cysts. The incidence is highest in patients between the ages of ten and twenty years, and the location of the cyst is usually in the posterior body of the mandible.

II. MANDIBULAR TUMORS

Tumors of the mandible may be divided into four categories: odontogenic, nonodontogenic, those of indeterminate origin and malignant. The odontogenic type includes those that arise from dental germ, while the nonodontogenic group consists of tumors of the bone, cartilage, nerve and vascular constituents of the mandible that are not involved in odontogenesis. The indeterminate origin group contains lesions of speculative etiology, and those closely simulating mandibular tumors in appearance. The malignant tumor category encompasses the primary malignant tumors, metastases and tumors involving the mandible by direct extension from the surrounding soft tissues.

In general, the roentgenographic appearance of benign and malignant tumors of the mandible is similar to that of these tumors when they occur in other bones in the body. The margins of benign tumors are usually well defined;[15] these tumors have an intact cortex even though it may be thinned and expanded; and they more frequently cause resorption of tooth roots, possibly attributable to their comparatively slow growth rate. Malignant tumors, of course, are the antithesis of the benign tumors, and, in addition, may have a periosteal reaction or "sunburst" appearance.

A. ODONTOGENIC TUMORS

These tumors arise from the cells involved in odontogenesis. On microscopic examination, their cytologic pattern suggests an origin from mesoderm, ectoderm or both. Some have a roentgenographic appearance of diagnostic significance, notably the "calcified odontomas," the encapsulation of which in most cases is visualized as a thin radiolucent area at the circumference of the tumor.[15]

1. *Ameloblastoma or Adamantinoma.* Histologically, these tumors show wide variations but in practically all cases cells resembling the stellate reticulum of the enamel organ can be identified.[17] Stafne[15] believes that they arise from ectodermal cells in enamel organ, periodontal membrane, follicle, or in the epithelial lining of dentigerous cysts.

An alternate theory is that ameloblastomas arise from the "debris of Malassez,"[17] or cells that originally were in the tubular enamel structure called Hertwig's sheath. This sheath forms the tooth roots and then disintegrates; any remaining cells will then lie in the periodontal membrane as the "debris of Malassez."[13]

Clinically, they produce painless expansion of the mandible in young adults. The frequency is somewhat higher in the Negro race. The site of origin is frequently in the angle of the mandible with extension into the ramus (Fig. 7).[14]

The roentgenographic appearance is varied. Ameloblastomas may be monolocular

and may resemble any type of cyst. A more typical appearance is that of multiple small radiolucent areas, which are variable in size and simulate a cluster of grapes (Fig. 8, *A*, *B* and *C*). These areas are separated by distinct septa.[16] The cortex usually is only expanded but destruction and invasion of the soft tissues may be present.

Jaffe[7] believes that these tumors are benign but may recur or occasionally metastasize. Robbins,[13] on the other hand, questions whether adamantinomas are malignant even though some metastases have been claimed. He believes that they are locally destructive benign tumors which tend to recur following incomplete surgical treatment.

2. *Odontoma.* Odontomas are considered to contain all the elements of dental structure, such as dentin, enamel, pulp, and cement. They occur in the area in which there has been failure of formation of a normal tooth. Usually, they contain variable amounts of calcification.

An important roentgenographic finding is a radiolucent area of varying width surrounding the tumor. This is due to a fibrous capsule separating the tumor from adjacent structures.

Odontomas as a group are called "composite odontomas,"[13] which are further divided into complex and compound types. The complex composite odontoma is roentgenographically seen as a mass of irregular calcification in a globular arrangement with a distinct separation from the surrounding bone (Fig. 9). There is no resemblance to actual tooth formation. The compound composite odontoma differs from the former in that it contains calcified dental tissue that actually resembles small teeth (Fig. 10), the number being variable. Either type may expand the mandibular cortex.

3. *Cementoma.* Cementomas arise from the periodontal membrane of fully erupted teeth. The membrane proliferates into fibrous connective tissue that calcifies. Because of the hyperplasia of fibrous tissue, the early roentgenographic appearance is

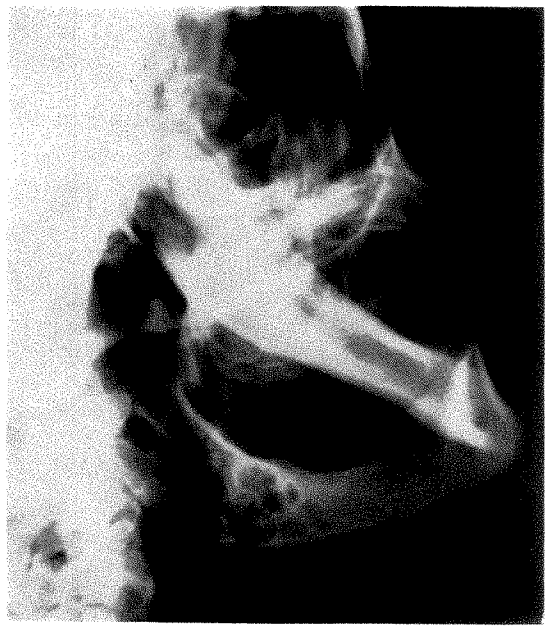


FIG. 7. A thirty-three year old woman with a one year history of sharp pain in the left jaw, radiating to the temple. At operation the pathologic diagnosis was ameloblastoma.

that of a radiolucent cavity which progressively changes to a characteristic calcified mass *attached* to the tooth root and surrounded by a small radiolucent cavity.[16,15] These relatively frequent tumors are not diagnosed often because they are usually asymptomatic. They occur in persons from twenty to sixty years of age and are slightly more common in women and in Negroes.[3]

4. *Enameloma.* These lesions possibly arise from remnants of the enamel anlage cells. They are rare and appear roentgenographically as small opaque tumors at the bifurcation of the tooth roots. They are smaller than odontomas and more radiopaque than cementomas.[3]

5. *Myxoma.* These tumors arise from the mesenchymal portion of the dental germ and histologically contain myxomatous connective tissue.[15] The roentgenographic appearance is varied but may simulate that of dentigerous cysts by being associated with unerupted teeth. In some instances myxomas are found in an area where there is congenital absence of teeth; this supports the contention that these tumors differ

from other myxomas and are odontogenic in origin.[17] Myxomas are partially encapsulated, and when large, give a "honeycombed" appearance which simulates an ameloblastoma.

6. *Fibroma.* Histologically, fibromas contain a few ectodermal cells and occasionally some calcification;[15] however, they arise from the mesenchymal portion of the dental germ. Roentgenographically, they appear as relatively well-defined radiolucent areas in close apposition to the crown or root of

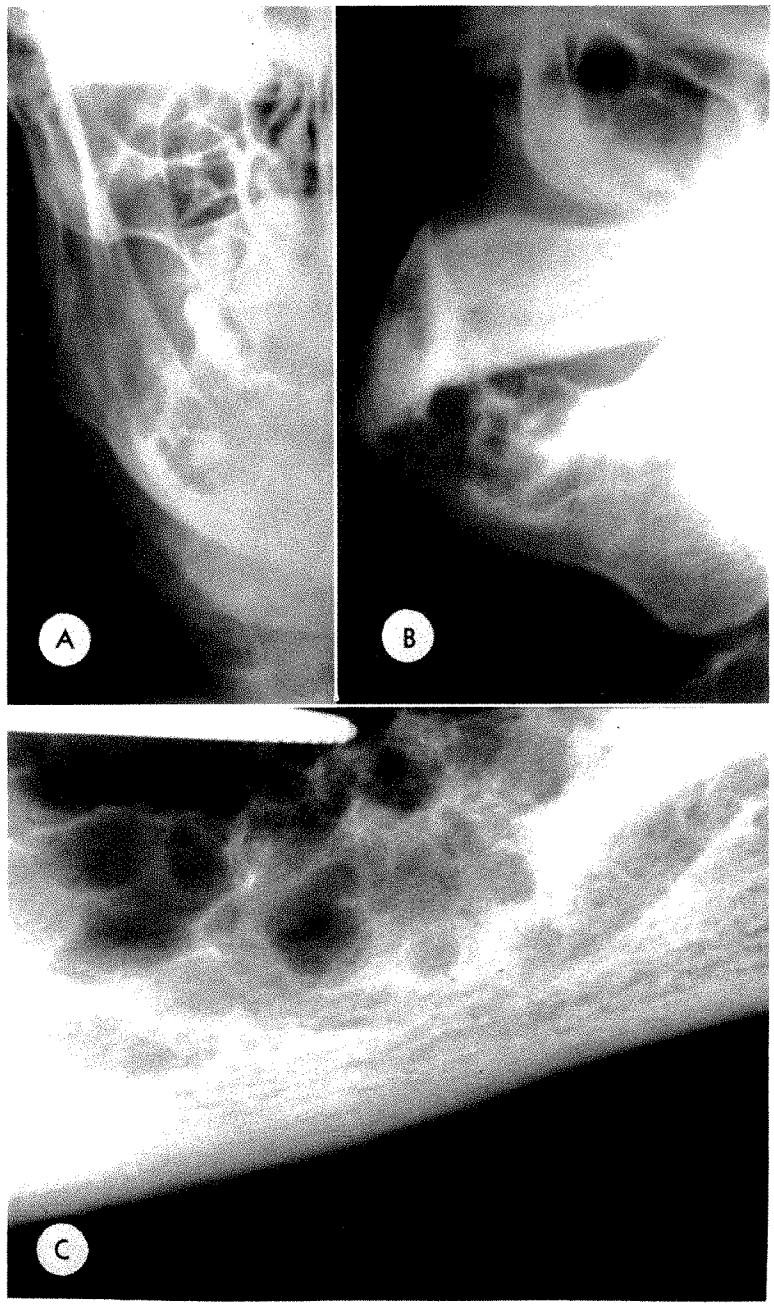


FIG. 8. (*A, B* and *C*) A seventy-two year old woman with a fifteen year history of asymptomatic swelling of the right mandible that gradually increased in size. The pathologic diagnosis from a specimen removed at operation was ameloblastoma.

the tooth, thus simulating radicular or dentigerous cysts. These tumors are classified by some authors as a variant of fibrous dysplasia.

B. NONODONTOGENIC TUMORS

These tumors are histologically similar to tumors of nerve, vascular, bone and cartilaginous tissues elsewhere in the body. Roentgenographically, the criteria for diagnosis are much the same when these tumors involve the mandible.

1. *Osteoma.* This is a rare benign tumor that is seen at the angle of the mandible, originating in the subperiosteal layers without invading the medullary bone.[10] The tumor may be on a pedicle or have a wide base,[15] with a density varying from very opaque to that of normal bone. Trabeculae may be seen to extend from the surrounding bone into the tumor; occasionally, the margins are corticated.[14]

2. *Osteoid Osteoma.* The majority of these tumors are cortical or just subcortical. Local pain is a prominent feature when they occur in the mandible as it is in other bones. The roentgenographic picture is that of a sclerotic nidus in a radiolucent area. The surrounding bone appears more dense than normally.[15]



FIG. 9. A thirty-six year old man with a three week history of swelling of the left side of the face. There was no numbness but he had some local pain. At operation the diagnosis of complex composite odontoma was confirmed. This is an unusually large lesion. Note the clear space surrounding the tumor, indicating encapsulation.



FIG. 10. A twenty-nine year old man in whom a compound composite odontoma was an incidental finding on routine dental survey roentgenograms. The patient had no symptoms referable to the mandible.

3. *Chondroma.* Chondromas are tumors that are primarily cartilage but because of osteoblastic activity may evolve into an osteochondroma or osteoma. The roentgenographic appearance is that of a cortical, raised radiolucent tumor with irregular areas of calcifications. Even though there may be a well-defined sclerotic rim, these tumors should be considered potentially malignant.

4. *Neurofibroma.* These tumors present a complex histopathologic picture. For the radiologist's purpose, they may be considered to be benign tumors that originate from the sheath of a peripheral nerve. In the mandible they characteristically arise in one of two locations. The first is in the mandibular canal, where the tumor will produce an oval expansion or radiolucent area with a fairly well-defined periphery.[15] This tumor is actually a fusiform expansion of the mandibular branch of the fifth cra-

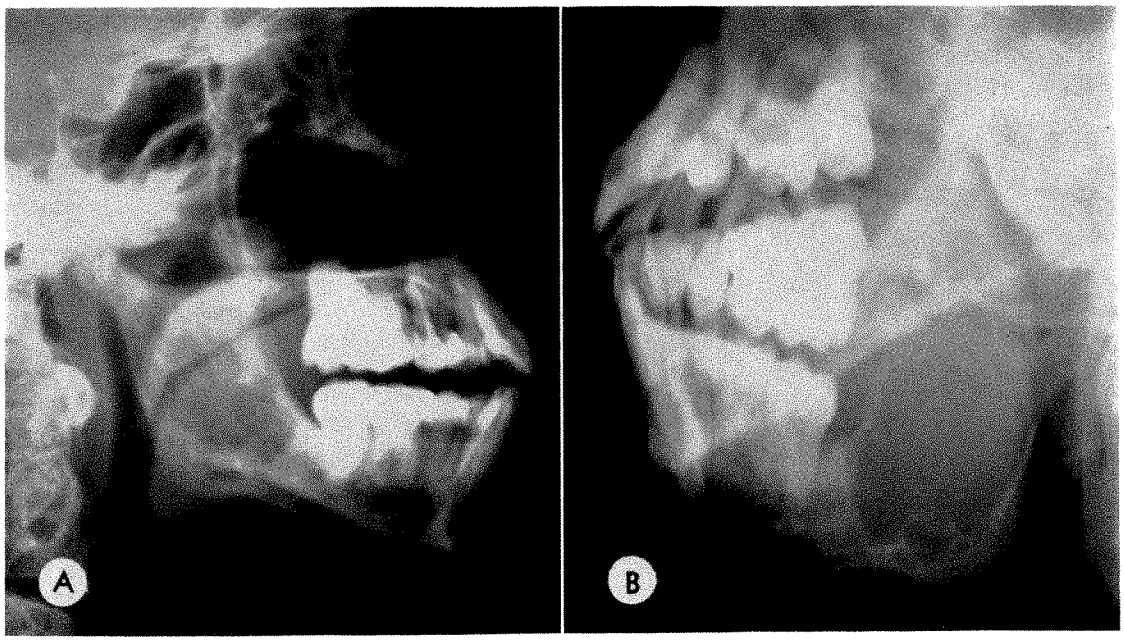


Fig. 11. (*A* and *B*) A twenty year old female with a one year history of a painful swelling of the upper gum. Roentgen examination of the mandible showed multiple osteolytic areas. Several teeth were loose. The serum calcium was 19.3 mg. per 100 ml., the serum phosphorus, 3.1 mg. per 100 ml. Needle biopsy specimens of the mandible, showing numerous giant cells, were consistent histologically with the diagnosis of hyperparathyroidism. A functional parathyroid adenoma was removed surgically.

nial nerve. The second site of development is in one of the periosteal nerves. As the tumor enlarges, it will cause erosion of the adjacent bone with formation of a pitlike area. It may have a thin shell of bone covering the tissue, thus giving it a cystic appearance.[10]

5. *Hemangioma.* Hemangiomas are vascular tumors that contain varied amounts of connective tissue stroma and in bone commonly arise in the spongiosa. The two histologic types are capillary and cavernous, both being rare. Clinically, they may produce bone expansion or pain. Of the few reported in the medical literature, the majority have been in young adults. If, on roentgenograms, coarse trabeculae can be seen to extend at right angles from the surrounding bone into the lesion, Stafne[15] feels that this is pathognomonic of hemangiomas. Pizer[11] reports a case of cavernous hemangioma in which the tumor appeared as an ill-defined radiolucent area without expansion. In another case of cavernous hemangioma,[2] expansion of the cortex with coarse long bony trabeculae was demonstrated suggesting an osteogenic as well as an osteolytic process. This hemangioma showed the more typical bony characteristics which occur when this tumor involves the vertebral bodies.

C. TUMORS OF INDETERMINATE ORIGIN

As the term implies, these entities do not have a clear etiopathogenesis; they may, in fact, not represent true tumors. However their roentgenographic appearance so closely resembles that of tumors that they are included as "tumors of indeterminate origin."

1. *Giant Cell Tumors.* These tumors microscopically are shown to contain multinuclear giant cells, a feature shared with several other tumors. Jaffe[7] believes that most of the previously diagnosed giant cell tumors of the mandible were either the "brown tumor" of hyperparathyroidism (Fig. 11, *A* and *B*) or giant cell reparative granulomas that are a response to inflammation. He feels that the neoplastic giant

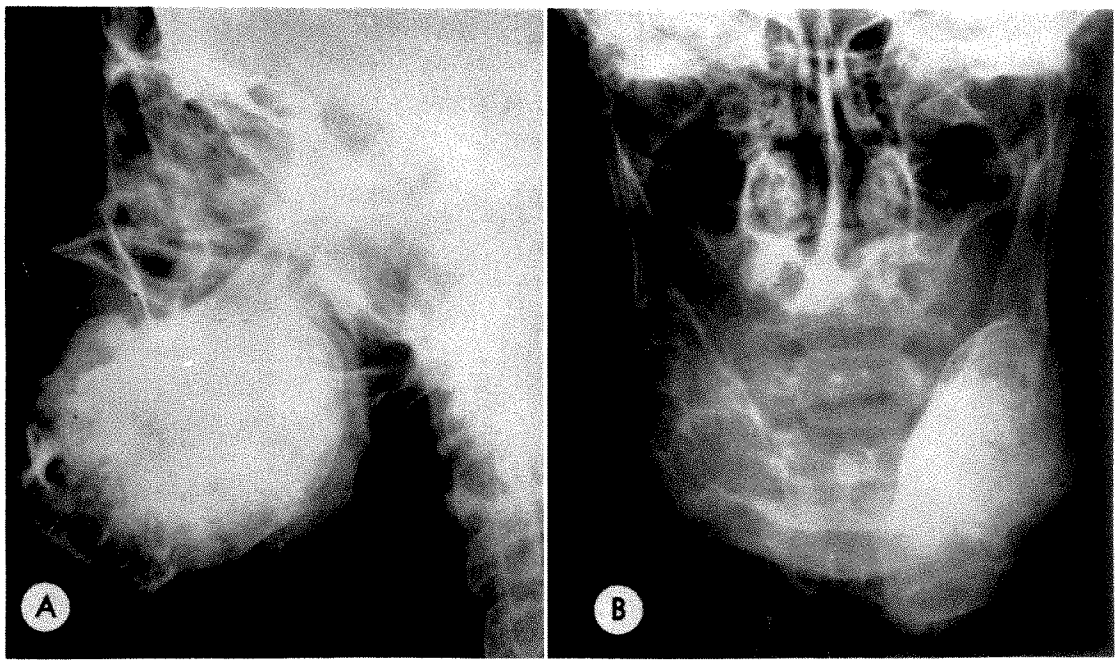


FIG. 12. (*A* and *B*) A fifty-three year old woman with bilateral mandibular swellings for seventeen years. The pathologic diagnosis was osteogenic fibroma.

cell tumor seen in other bones is exceedingly rare in the mandible. Roentgenographically, mandibular giant cell tumors have an extremely varied appearance. They may appear either as a single area of destruction with no septa or as multilocular lesions with septa that are more numerous and not as distinct and regular as those in ameloblastomas.[16] Jaw expansion is common.[14]

According to Jaffe[7] giant cell reparative granulomas occur between the ages of ten and twenty-five years, while giant cell tumors are found in patients over twenty years of age.

2. *Fibrous Dysplasia.* The classification of the fibro-osseous lesions is a difficult task. The term "fibrous dysplasia" has been used by many authors with various meanings. Some classify osteofibromas (Fig. 12, *A* and *B*), ossifying fibromas, and fibro-osseous dysplasia as variants of fibrous dysplasia. Zimmerman, Dahlin, and Stafne[19] describe fibrous dysplasia histologically as a tumor of connective tissue with active osseous metaplasia. They believe that it is a benign fibro-osseous process that involves the spongiosa of the bone causing expansion and deformity. It is a disease of unknown etiopathogenesis, thought to be more common in persons in the second and third decades of life, with approximately 70 per cent of the cases in females.[19] The roentgenographic appearance may be quite varied—from a unilocular well-defined lesion (Fig. 13) (monostotic fibrous dysplasia) to a multilocular mottled and sclerotic lesion (polyostotic fibrous dysplasia). Both the unilocular and the multilocular types often contain poorly defined sclerotic areas that give an over-all appearance of "smudging," which is almost pathognomonic (Fig. 14). It rarely may appear as a very radiopaque tumor with irregular margins and may become almost imperceptibly fused with the bone.

Caffey and Williams[1] believe that familial fibrous swelling of the jaws is a separate entity and should be classified as such. They attribute the term "*familial* fibrous dysplasia" to Dr. W. H. Jones, who coined the words "cherubism" and "eyes raised to heaven" look. Cherubism produces a mas-

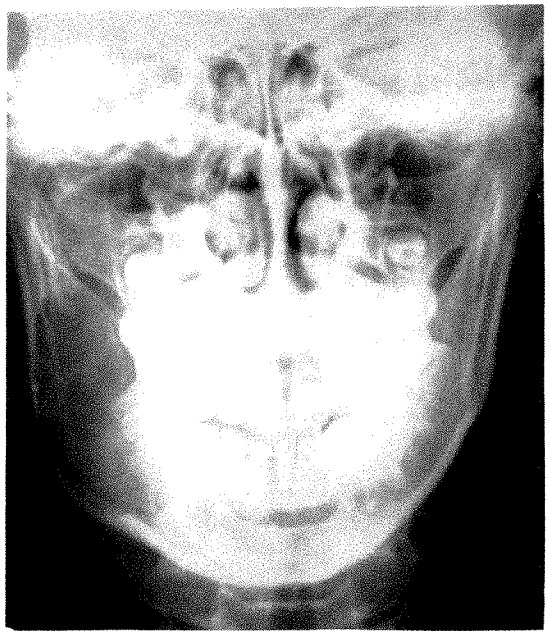


FIG. 13. A twelve year old girl with an asymptomatic swelling of the right jaw for eight days. The pathologic diagnosis was fibrous dysplasia. Note the monolocular appearance and the "smudging."



FIG. 14. A forty-eight year old man who had had a partial nasal obstruction at twelve years of age, after which all of the bones of the left side of the forehead and face increased in size. Serum calcium, phosphorus and phosphatase levels were normal. The diagnosis was fibrous dysplasia.

sive bilateral swelling of the cheeks and jaws which stretches the skin, pulling on the lower eyelids and giving the "eyes raised to heaven" look. It begins in the first three years of life.[10] The roentgenographic appearance is that of bilateral multilocular expansion of the mandible with thinning and expansion of the cortex (Fig. 15; and 16, *A*, *B* and *C*). The swelling is firm and painless, and improves markedly after puberty. In some cases there is almost complete restoration of the normal features.

3. *Reticuloendotheliosis.* The term "reticuloendotheliosis" includes the lesions of Letterer-Siwe disease, Hand-Schüller-Christian disease and eosinophilic granuloma. Pugh[12] considers all three to be variations of the same disease but of different severity of involvement. The roentgenographic appearances in the mandible are similar: numerous rather sharply defined but not corticated radiolucent areas. The characteristic picture is that of a sharply outlined destruction of the alveolar ridge with the unusual finding of teeth that are not imbedded in bone but appear to be "floating in air" (Fig. 17, *A* and *B*; and 18).

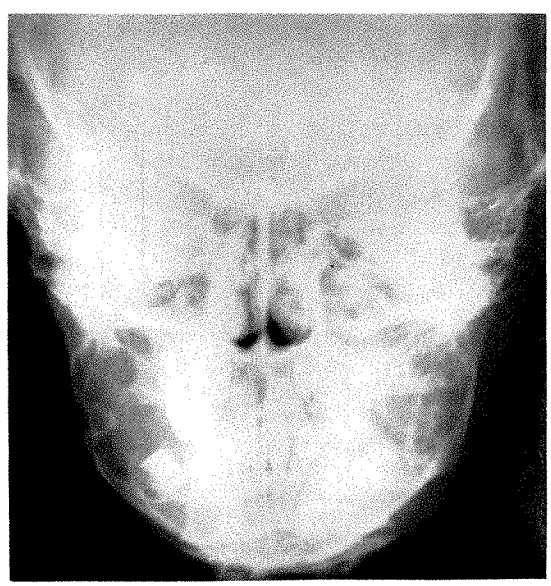


FIG. 15. An eight year old boy with an asymptomatic bilateral swelling of the jaw for three years. All siblings were normal. The pathologic diagnosis was fibrous dysplasia (cherubism). Note the bilateral cystic involvement.

4. *Infantile Cortical Hyperostosis.* This disease is a self-limiting process of obscure etiology which appears between the ages of three and six months. Clinically, the infants have fever, irritability, leukocytosis and anemia. Roentgenographically, the mandible, ribs, clavicles, and tubular bones show subperiosteal bone lamination with some cortical thickening.[18]

D. MALIGNANT TUMORS

Carcinomas are unusual and probably always arise from coexisting abnormalities. For example, Figure 19 shows a muco-epidermoid cyst that, following partial surgical removal, recurred as a muco-epidermoid carcinoma.

Malignant tumors of vascular and neurogenic origin do occur in the mandible but are exceedingly uncommon. Tumors of the marrow or hematopoietic system are more

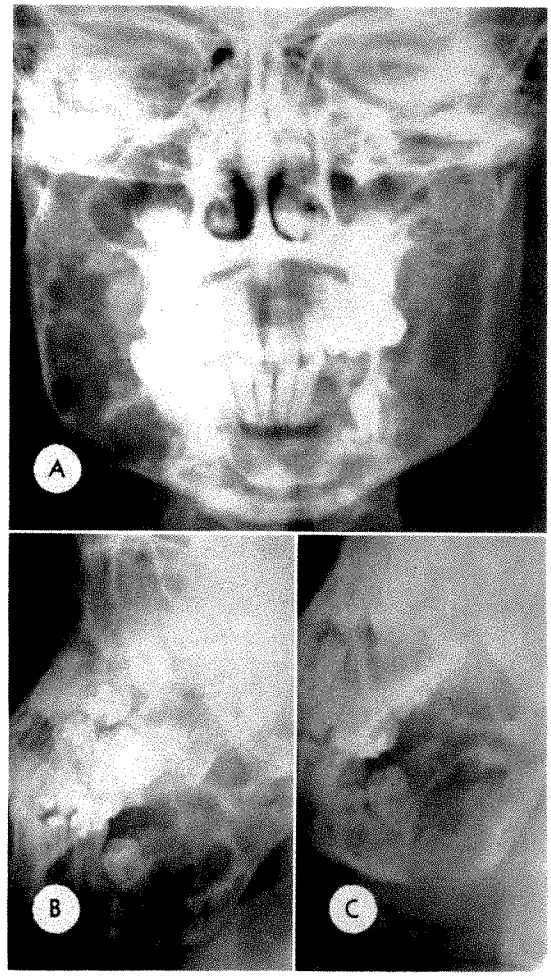


FIG. 16. (*A*, *B* and *C*) A ten year old boy who was never noticed to have mandibular swelling until the local physician called it to the attention of the parents. The patient was asymptomatic and all siblings were normal. The pathologic diagnosis was fibrous dysplasia (cherubism). Note that the involvement is bilateral.

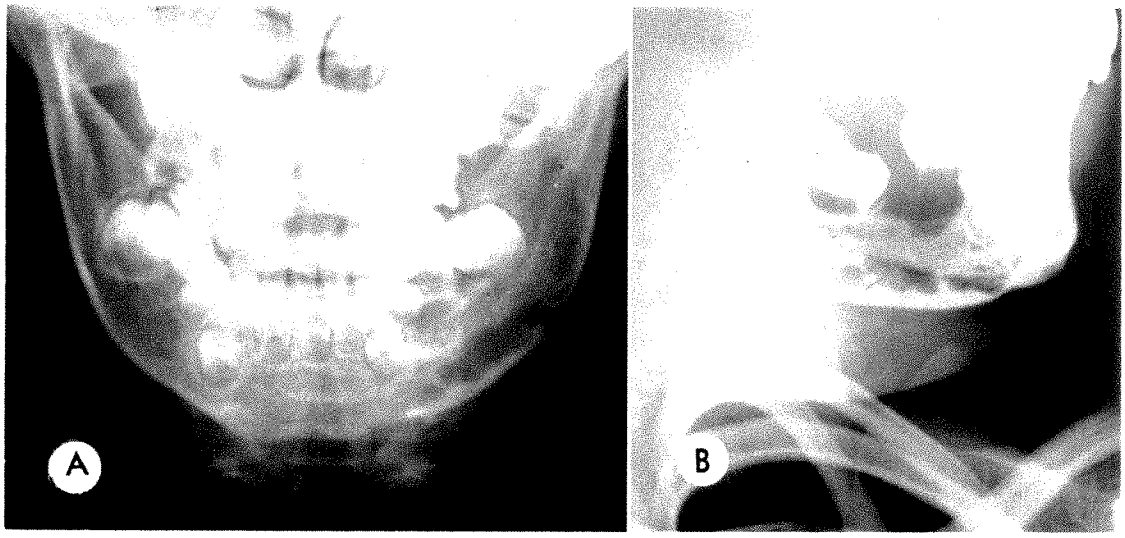


FIG. 17. (*A* and *B*) A three and one-half year old girl was healthy until one year of age when she developed a lump behind the left ear, with an associated yellowish dermatitis of the scalp and diabetes insipidus. Biopsy of the osteolytic lesions of the skull showed Hand-Schüller-Christian disease.



FIG. 18. A sixteen month old girl had swelling of the left jaw for three weeks which continued to increase in size. The child cried when the area was touched. Biopsy of the mandible showed eosinophilic granuloma. A later biopsy of the skin of the abdomen showed changes compatible with Letterer-Siwe disease.

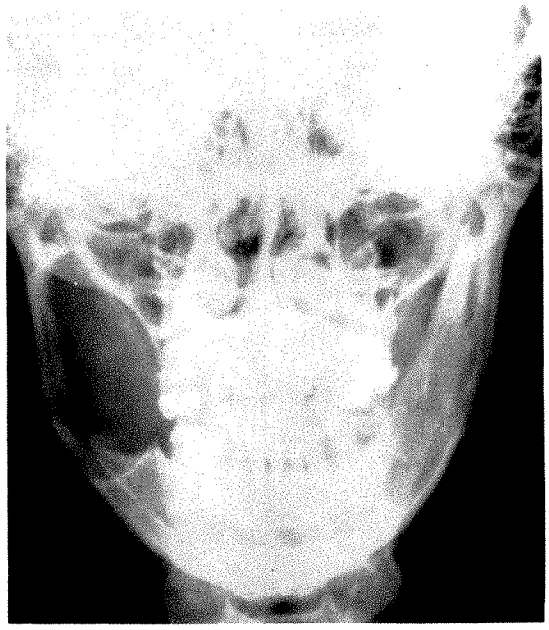


FIG. 19. A thirty-six year old woman who had pain and aching in the right temporal area for five months at the time this roentgenogram was made. The cyst was curetted and the pathologic diagnosis of the tissue was muco-epidermoid cyst. Five years later the cyst recurred and a biopsy specimen at that time showed muco-epidermoid carcinoma.

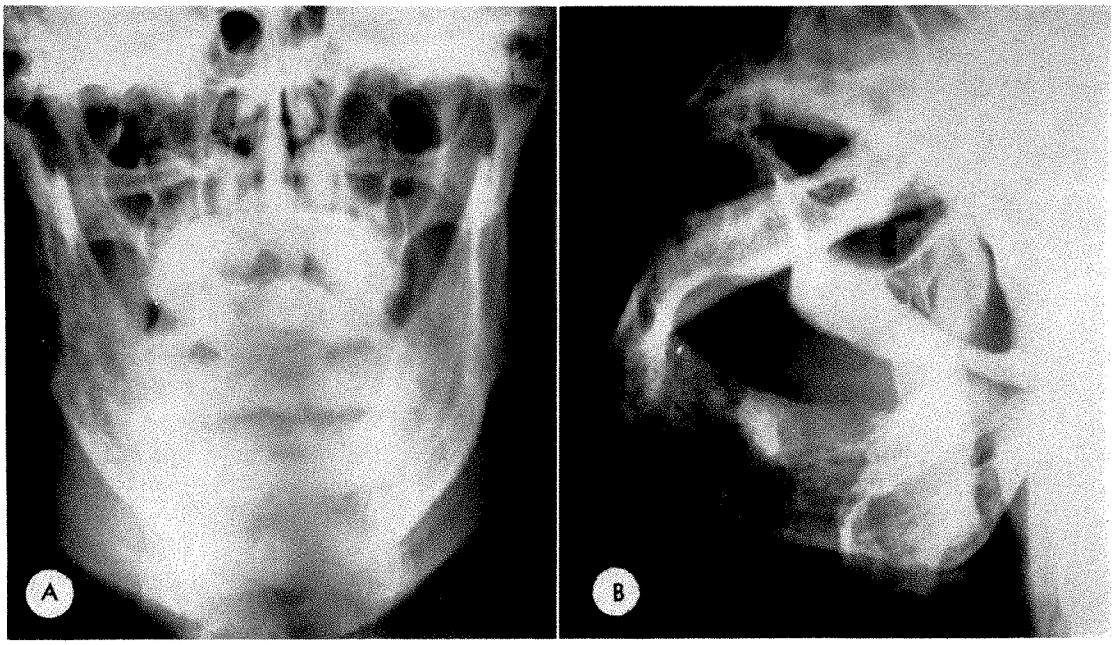


FIG. 20. (*A* and *B*) A forty year old man with pain in the right mandible for one year. Previous treatment consisted of dental extractions and penicillin. The pathologic diagnosis was fibrosarcoma. Note the soft tissue swelling, the fine perpendicular periosteal spicules, and the osteolytic involvement of the body of the mandible.

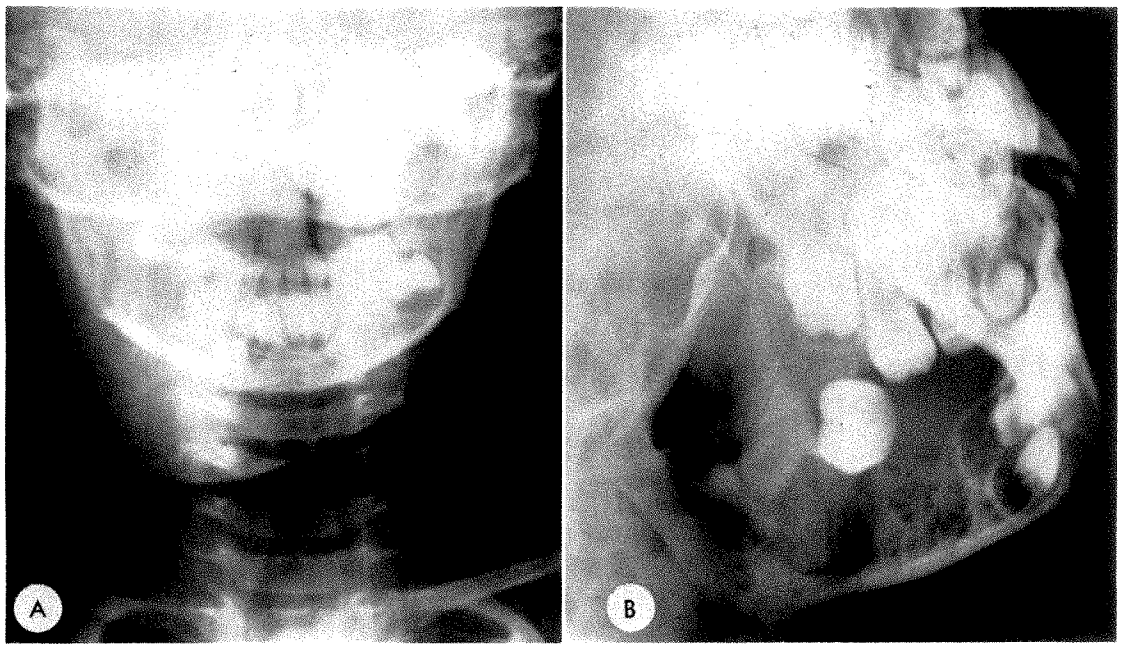


FIG. 21. (*A* and *B*) A three and one-half year old girl had slight swelling over the right lower gum for four weeks which gradually increased in size. Biopsy showed Ewing's sarcoma.

frequent. Because of the embryonic origin of the mandible, sarcomas comprise a large part of the rare primary malignant tumors.

1. *Primary Malignant Tumors.* Primary malignant tumors of the mandible are rare but do occur. Osteogenic sarcoma, Ewing's sarcoma, chondrosarcoma, reticulum cell sarcoma, lymphosarcoma, and fibrosarcoma (Fig. 20, *A* and *B*) are the principal types. As in other bones these tumors produce irregular ill-defined, areas of bone destruction. The margins are indistinct and fade into the surrounding bone (Fig. 21, *A* and *B*). Osteogenic sarcomas[10] and reticulum cell sarcoma may have mixed osteolytic and osteoblastic components.

Osteogenic sarcomas may cause expansion of the cortex with periosteal new bone

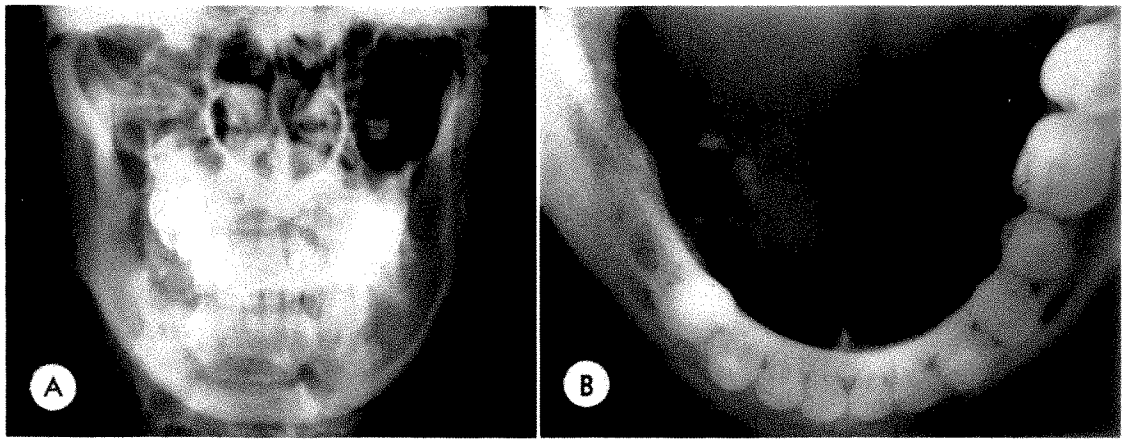


FIG. 22. (*A* and *B*) An eighteen year old woman with a history of a rapidly growing lump on the right lower gum. Just prior to admission she noticed some anesthesia of the lip and teeth. The diagnosis based on biopsy at resection of the mandible was osteogenic sarcoma. Note the soft tissue mass and perpendicular spicules.

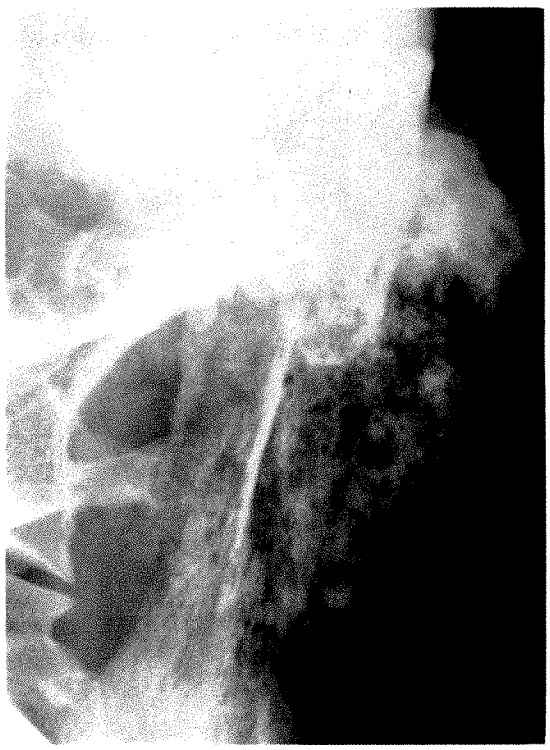


Fig. 23. A fifty-four year old man with a firm fixed nodule anterior to the left ear for ten years. Treatment consisted of rubbing with a bland salve and, during the past year, 20 deep radiation treatments were given after a presumptive diagnosis of parotid tumor. Surgical pathologic diagnosis was osteochondrosarcoma.

formation which also occurs in Ewing's sarcoma; however, perpendicular spicules or striations are more common (Fig. 22, *A* and *B*).

Gerry and Williams[4] reviewed the literature in 1955 and found only 6 cases besides the 2 of their own in which the reticulum cell sarcoma was primary in the mandible. In these cases, it was encountered more often in men than in women, and occurred up to the age of forty. All 8 patients had local pain but also had an unusual over-all general feeling of well-being.

Chondrosarcomas are thought to arise from cartilaginous arrests trapped in the mandible[5] or from osteochondromas (Fig. 23). Pain is common and distant metastases occur late.

Multiple myeloma produces a roentgenographic picture of diffuse irregular areas of destruction of bone with erosion of the tooth roots when they are in close proximity. At times, these tumors resemble the more typical "punched out" lesions that occur in involvement of other bones (Fig. 24, *A* and *B*). They are most frequently found in the middle-age group with more men than women affected.[12] Areas of bone destruction without cortication and away from the apex of a tooth should arouse suspicion of this diagnosis. These tumors destroy and perforate the cortex and several small lesions may coalesce to form large lesions. Stafne[15] suggests that when myeloma involves the mandible the cortex will not be expanded.

2. *Metastases.* Metastases to the mandible occur late in malignant diseases, usually after involvement of other bones. The tumors that frequently metastasize to the mandible are those of breast (Fig. 25, *A* and *B*), thyroid, lung, kidney and prostate. Occasionally, head and neck tumors, carcinoma of the gastrointestinal tract, carcinoma of the cervix and vagina, lymphosarcomas and melanomas may metastasize to the mandible.[17]

Roentgenographically, metastases appear as solitary or multiple irregular radiolucent defects. In some tumors, such as those of the prostate or lymphosarcoma, the metastatic area will have a sclerotic component. In any irregular defect with a poorly defined margin, metastasis should be a cardinal differential diagnostic consideration.

3. *Extension from Surrounding Tissues.* Any tumor of the face or oral cavity may extend to involve the mandible. The degree of involvement may vary from a pressure defect in the cortex to actual perforation of the cortex and medullary invasion.

DIFFERENTIAL DIAGNOSIS

The final diagnosis of a mandibular lesion should be based upon pathologic examination. Some lesions can be identified by clinical and roentgenographic methods. The rare malignant tumors present such varied features that a pathologic specimen is essential for positive identification.

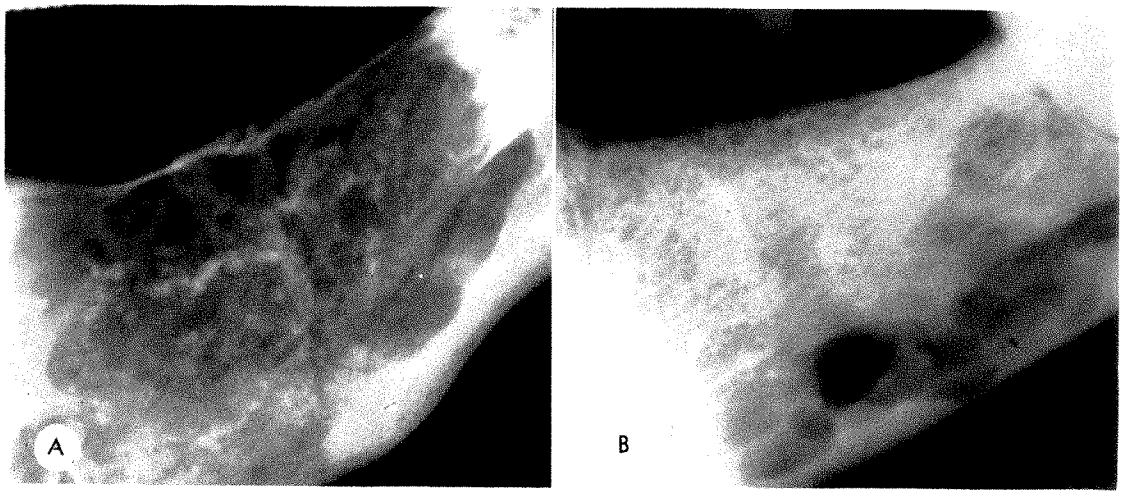


FIG. 24. (*A* and *B*) A sixty-six year old man had rib pain for three months. There were widespread radiolucent defects of bone. Sternal marrow aspirate was diagnostic of multiple myeloma.

To differentiate lesions of the mandible roentgenographically and to classify them tentatively, these questions must be answered and the findings must be correlated:

(1) Is the lesion unilocular, multilocular, or multicystic?

(2) Are the borders of the lesion well defined or diffuse and hazy?

(3) What is the location and extent of involvement?

(4) Is the cortex expanded or destroyed?

(5) Does the involved portion contain calcific or ossifying areas?

(6) Is there periosteal reaction or a soft tissue mass?

(7) Are other bones involved?

A single cystic lesion with a well-defined margin that has no relationship to a tooth is most frequently a squamous cyst. If it is oriented to the root of a tooth, then the

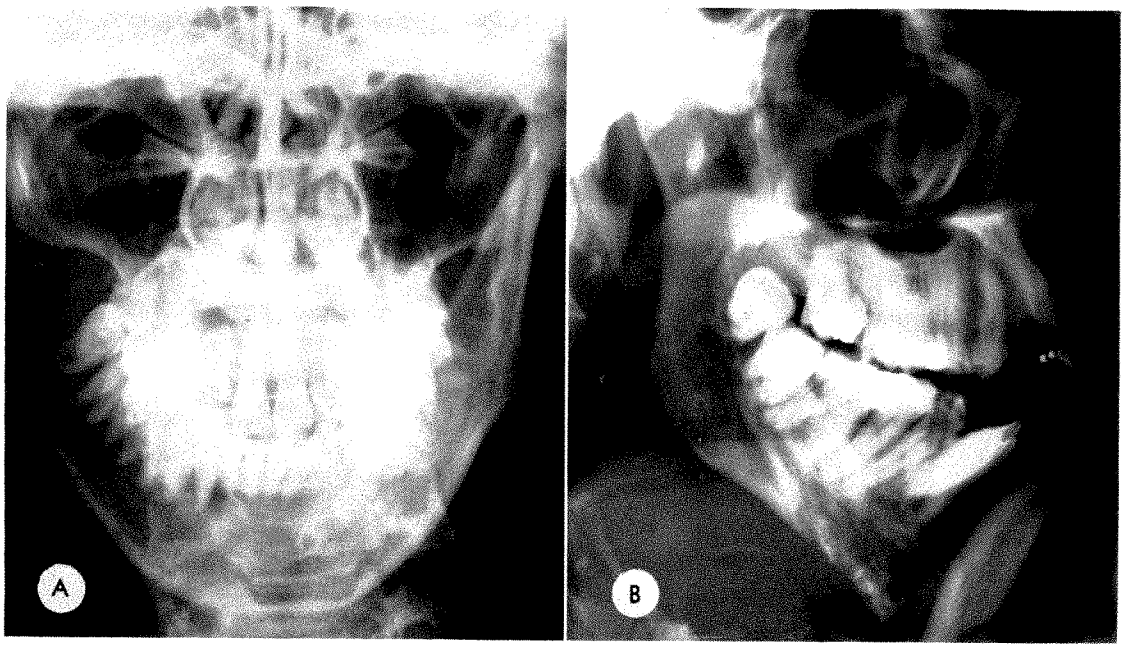


FIG. 25. (*A* and *B*) A thirty-three year old woman who had a right radical mastectomy for scirrhous carcinoma of the breast. Note the complete destruction of the cortex by an osteolytic process.

possibility of a radicular cyst should be considered. When a cystic lesion is oriented to the crown or contains a tooth, it is probably a dentigerous cyst. Fibromas and myxomas are tumors that may simulate dentigerous or radicular cysts. Infrequently, fibrous dysplasia may be seen roentgenographically as a single monolocular lesion. Neurofibromas are observed as an oval radiolucency aligned to the central mandibular canal or in the subperiosteal area.

Squamous cysts may have a multilocular well-defined appearance on initial roentgenographic study, but additional roentgenography will often reveal a single cyst with lobulated margins.

Fibrous dysplasia commonly has a multilocular appearance with gross expansion of the cortex; the "smudged" appearance due to osseous metaplasia is characteristic if present. Fibrous dysplasia is frequently unilateral while cherubism is bilateral and occurs in a younger age group.

A difficult differential diagnostic problem arises when there are multiple well-defined cystic areas. Ameloblastomas have varied appearances but the typical one is of multiple small radiolucencies arranged in discrete clusters, like grapes. The compartments are usually rounder than those of the giant cell tumors, and the trabeculae of the giant cell reparative granulomas or of the tumors of hyperparathyroidism are more numerous and not as distinct as those of ameloblastomas. To distinguish between giant cell granulomas and hyperparathyroidism, it is necessary to review the clinical and laboratory data. Defects of reticuloendotheliosis do not occur in clusters and rarely affect *only* the mandible. Their most characteristic location is in the alveolar process with destruction and the appearance of "floating teeth."

Single or multiple radiolucent defects with poorly defined margins should always suggest metastases (Fig. 23). It is here that care should be taken to differentiate between metastases, myeloma, and primary malignant tumors. Biopsy is indicated if the diagnosis is not conclusive from the clinical and laboratory data. Primary malignant tumors may mimic any lesion but the roentgenographic characteristics of malignant tumors of the mandible are the same as of other bones. Irregular osteolytic or osteosclerotic areas with periosteal reaction are typical.

The odontogenic tumors show areas of calcification surrounded by a radiolucent area of varying thickness which indicates encapsulation. If the calcifications are grossly irregular or toothlike, the tumor is an odontoma. Cementomas are attached to a tooth root while enamelomas are more radiopaque and appear at the bifurcation of the tooth roots.

Osteomas of the mandible are rare; they usually occur at the angle of the mandible and roentgenographically appear as a dense tumor on a pedicle. Osteoid osteomas have the typical appearance of a sclerotic area with a central nidus. Infantile cortical hyperostosis is diagnosed on the basis of the age of the patient, his clinical history, and the roentgenographic finding of thickened lamellated subperiosteal bone. Infrequently, fibrous dysplasia may appear roentgenographically as a dense irregular sclerosis with indistinct margins.

Tumors that involve the mandible by direct extension from the surrounding tissues are usually recognized clinically and the mandible is examined roentgenographically to confirm bone involvement. In some rare borderline cases, however, a biopsy must be performed to distinguish between a mandibular tumor with soft tissue extension and a primary soft tissue tumor with mandibular involvement.

While not included in this discussion, osteomyelitis and radiation necrosis should be considered in the differential diagnosis, although the clinical history will usually eliminate these entities.

CONCLUSION

1. Some cysts and tumors may be diagnosed from the roentgenograms with reasonable accuracy.

2. In most cases, however, a biopsy is almost mandatory because of the extremely variable roentgenographic features of mandibular cysts and tumors.

3. Roentgenographic findings of mandibular cysts and tumors are presented to aid in the differential diagnosis.

F. W. O'Brien, Jr., M.D.
Cleveland Clinic
2020 East 93rd Street
Cleveland 6, Ohio

The authors wish to thank Dr. Lawrence J. McCormack of the Department of Anatomic Pathology for his constructive comments on this paper.

REFERENCES

1. Caffey, J., and Williams, J. L. Familial fibrous swelling of jaws. *Radiology*, 1951, *56*, 1–14.
2. Case records of the Massachusetts General Hospital; Case 42021. *New England J. Med.*, 1956, *254*, 70–73.
3. de Lorimier, A. A., Moehring, H. G., and Hannan, J. R. Clinical Roentgenology. Volume II: The Head, Neck and Spinal Column. Charles C Thomas, Publisher, Springfield, Ill., 1954, pp. 272–319.
4. Gerry, R. G., and Williams, S. F. Primary reticulum-cell sarcoma of mandible. *Oral Surg.*, 1955, *8*, 568–581.
5. Geschickter, C. F., and Copeland, M. M. Tumors of Bone. Third edition. J. B. Lippincott Company, Philadelphia, 1949, pp. 654–685.
6. Gray, H. Anatomy of the Human Body. Twenty-seventh edition. Lea & Febiger, Philadelphia, 1959, pp. 1225–1229.
7. Jaffe, H. L. Tumors and Tumorous Conditions of the Bones and Joints. Lea & Febiger, Philadelphia, 1958, pp. 425–450.
8. Pancoast, H. K., Pendergrass, E. P., and Schaeffer, J. P. The Head and Neck in Roentgen Diagnosis. Charles C Thomas, Publisher, Springfield, Ill., 1940, p. 318.
9. Patten, B. M. Human Embryology. Second edition. Blakiston Company, New York, 1953, p. 442.
10. Pendergrass, E. P., Schaeffer, J. P., and Hodes, P. J. The Head and Neck in Roentgen Diagnosis. Second edition. Volume I. Charles C Thomas, Publisher, Springfield, Ill., 1956, pp. 485–525.
11. Pizer, M. E. Cavernous hemangioma occurring in body of mandible. *Oral Surg.*, 1954, *7*, 1201–1203.
12. Pugh, D. G. Roentgenologic Diagnosis of Diseases of Bones. Williams & Wilkins Company, Baltimore, 1951, pp. 372, 475–478.
13. Robbins, S. L. Textbook of Pathology, with Clinical Applications. W. B. Saunders Company, Philadelphia, 1957, pp. 700–711.
14. Shanks, S. C., and Kerley, P., Editors. A Text-Book of X-Ray Diagnosis, by British Authors. Third edition. Volume I. W. B. Saunders Company, Philadelphia, 1957, pp. 364–384.
15. Stafne, E. C. Oral Roentgenographic Diagnosis. W. B. Saunders Company, Philadelphia, 1958, pp. 141–219.
16. Stafne, E. C. Value of roentgenograms in diagnosis of tumors of jaws. *Oral Surg.*, 1953, *6*, 82–92.
17. Thoma, K. Oral Pathology. Fifth edition. C. V. Mosby Company, St. Louis, 1960, pp. 780–833, 1168–1347.
18. Thompson, H. C., and Tabris, J. A. Infantile cortical hyperostosis. *Oral Surg.*, 1958, *11*, 8–13.
19. Zimmerman, D. C., Dahlin, D. C., and Stafne, E. C. Fibrous dysplasia of maxilla and mandible. *Oral Surg.*, 1958, *11*, 55–68.

THE RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ORAL ROENTGENOGRAPHIC FINDINGS*

By L. MANSON-HING, D.M.D.,† *and* E. CHERASKIN, M.D., D.M.D.‡
BIRMINGHAM, ALABAMA

A SURVEY of the literature shows a tendency of many investigators to associate the roentgenographic findings of alveolar bone loss and periodontal widening with the diabetic patient. There is implication of a linear relationship between these two findings and the diabetic state. In other words, the more pronounced the diabetic state (the higher the blood glucose) the more severe the alveolar bone loss and/or periodontal widening.

One report[1] has already analyzed the relation of oral roentgenographic findings with the glucose tolerance pattern. The evidence from that study indicates that there is a relationship between the glucose tolerance pattern and the presence of alveolar bone loss and periodontal widening.

This report will analyze the relationship of the two roentgenographic signs (*alveolar bone loss* and *periodontal widening*) to the fasting blood true glucose in a group of 100 routine dental patients.

REVIEW OF THE LITERATURE

Published material is available regarding the relationship between diabetes mellitus and alveolar bone loss and periodontal widening.

Alveolar Bone Loss. Many investigators have reported this finding as being associated with the diabetic condition.[4,5,8,10–12,18] One investigator[14] observed bone loss in 1 out of every 2 of a group of diabetic children. However, two teams[6,13] claim no alveolar bone loss. Sheridan *et al.*[15] observed normal alveolar bone in 24 per cent of patients with normal glucose tolerance patterns. They further observed that patients with increased or decreased glucose tolerance showed a greater incidence of alveolar bone loss and that most of the diabetic patients showed considerable bone loss in contrast to other groups of patients having more physiologic glucose tolerance patterns.

Periodontal Widening. Rutledge[14] observed this finding in 14 of 20 juvenile diabetics. The observations of Rudy and Cohen[13] on this finding are in agreement with those of Rutledge. However, Ziskin *et al.*[19] could not demonstrate periodontal widening in their series of cases. Sheridan *et al.*[15] reported that in patients with decreased glucose tolerance patterns, *i.e.*, persons who would commonly be called diabetic, there was a 62 per cent incidence of periodontal widening. These investigators also observed that the occurrence of this finding decreased with an increase in sugar tolerance.

METHOD OF INVESTIGATION

One hundred dental patients were studied in the Section on Oral Medicine at the University of Alabama School of Dentistry. As far as possible, the subjects were selected at random. Table 1 shows the age distribution of the group. It can be observed that the pattern approaches a typical unimodal curve. Included in this study were 22 males and 78 females.

The roentgenograms of each patient were studied for possible alveolar bone loss and periodontal widening. Alveolar bone loss was scored on a seven point scoring system where zero was the score given for a normal septal crest; a score of one was assigned for minimal alveolar bone loss; two for minimal to moderate alveolar bone loss; three for one-half the alveolar support missing; four for moderate to severe alveolar bone loss; five for no alveolar support;

* This investigation was supported in part by a traineeship grant (2G-15) from the Epidemiology and Biometry Section, Public Health Service and (A-2899) National Institute of Arthritis and Metabolic Diseases.
† Fellow in Oral Medicine, University of Alabama School of Dentistry, Birmingham, Alabama.
‡ Professor and Chairman, Section on Oral Medicine, University of Alabama School of Dentistry, Birmingham, Alabama.

and six for edentulous cases. Periodontal widening was scored on a four point scoring system where zero was the score given for no marginal widening; a score of one was given for minimal marginal widening; two for severe marginal widening; and three for edentulous cases.

A true glucose tolerance test was performed on each patient according to the methods of Somogyi[16,17] and Nelson.[9] No preparatory diet was recommended except for complete fasting for twelve hours prior to the laboratory examination. Complete details regarding the method of investigation are available in the publication by Sheridan *et al.*[15] For this study, only the information derived from the fasting blood glucose measurements was utilized.

RESULTS

The fasting blood glucose determination will be considered in the light of: (A) single roentgenographic signs, and (B) a combination of the two signs.

A. SINGLE SIGNS

An analysis of the data will be offered for: alveolar bone loss, and periodontal widening.

Alveolar Bone Loss. This roentgenographic sign will be discussed in terms of: (1) mean scores, (2) coefficients of variation, and (3) statistical significance. The scores of edentulous cases were included in this analysis since this condition would be an end result of alveolar bone loss. Although loss of teeth can be due to other causes, it is felt that the exclusion of these cases would be too discriminatory against those cases where the loss of teeth was due to alveolar bone loss. The available data do not permit the selection of those cases where loss of teeth was accompanied by severe alveolar bone loss.

TABLE I

AGE DISTRIBUTION

Age Group	Total No. of Patients
0– 9	1
10–19	9
20–29	19
30–39	27
40–49	19
50–59	17
60–69	8
Total	100

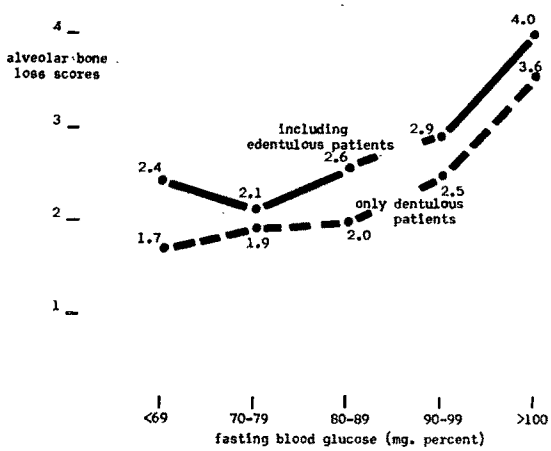


FIG. 1. Relationship of fasting blood glucose to alveolar bone loss.

1. Mean Scores. An examination of Figure 1 shows that alveolar bone loss was least in those patients with a fasting blood glucose of 70–79 mg. per cent. There was an increase in the scores calculated from patients having a fasting blood glucose of <69, 80–89, and 90–99 mg. per cent. There was a marked increase in alveolar bone loss in those patients with a fasting blood glucose of >100 mg. per cent. Figure 1 also shows a comparison with the mean scores obtained from dentulous cases alone. Basically, the curves follow the same pattern except in the <69 mg. per cent group. When the edentulous cases are excluded from this group, the mean score for alveolar bone loss is slightly less than the 70–79 mg. per cent group.

2. Coefficients of Variation. An examination of Table II reveals that basically the coefficients of variation decrease with an increase in the blood glucose percentages. The conclusion to be drawn is that the individual subjects seem to cluster about

TABLE II

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss Score	2.4	2.1	2.6	2.9	4.0
Standard Deviation	2.0	1.5	1.9	1.8	1.1
Coefficient of Variation	83	71	73	62	28
Mean Difference	0.3	0.5	0.3	1.1	
Standard Error of Difference	0.5	0.4	0.7	0.8	
Observed *t* Value	0.60	1.25	0.43	1.38	
Degrees of Freedom	50	71	43	15	
P	.5	>.1	.5	>.1	

the mean more in the upper blood glucose groups than in the lower ones.

3. Statistical Significance. From Table II it can be seen that there is no statistically significant difference between any of the blood glucose groups when compared to similar groups that are greater or less by 10 mg. per cent. This is evidenced by the probability factor of .5 and >.1. The P value was also checked for all the groups. There were no statistically significant differences shown except those between the >100 mg. per cent group and the <69, 70–79, and 80–89 mg. per cent groups. There the P values were <.05, <.005, and <.025, respectively. It is evident that the greatest significant difference occurs between the >100 mg. per cent group and the 70–79 mg. per cent group.

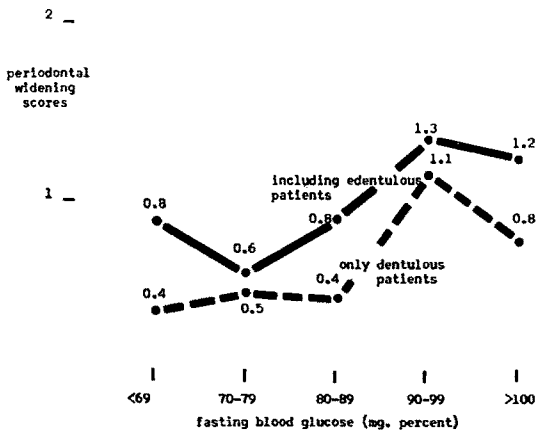


FIG. 2. Relationship of fasting blood glucose to periodontal widening.

Periodontal Widening. This roentgenographic sign will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance.

1. Mean Scores. Figure 2 shows the mean scores of all patients (dentulous and edentulous) and the mean scores when edentulous patients were excluded. An examination of these scores shows the effect of excluding the edentulous cases. Patients having severe periodontal widening would be scored but once, having lost their teeth, would not be counted and thus would lower the mean score of the blood glucose group they belong to. This effect is greatest in the <69 and >100 mg. per cent blood glucose groups. The rest of this analysis will pertain to the scores obtained from the study of all the patients. Figure 2 shows that periodontal widening is least in the 70–79 mg. per cent blood glucose group and, basically, this condition increases as the fasting blood glucose rises above or falls below that of this group.

2. Coefficients of Variation. An examination of Table III shows that the smallest coefficient of variation occurs at the 90–99 mg. per cent group and is greatest at the <69 group. In other words, the individual scores more closely cluster about the mean in the 90–99 mg. per cent group.

3. Statistical Significance. From Table

TABLE III

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO PERIODONTAL WIDENING

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Periodontal Widening Score	0.8	0.6	0.8	1.3	1.2
Standard Deviation	1.1	0.8	1.0	0.7	1.2
Coefficient of Variation	138	133	125	54	100
Mean Difference		0.2	0.2	0.5	0.1
Standard Error of Difference		0.32	0.22	0.28	0.54
Observed t Value		0.63	0.91	1.79	0.19
Degrees of Freedom		48	69	41	13
P		.5	>.2	>.05	.5

III it can be seen that there is no statistically significant difference between any of the blood glucose groups when compared to similar groups that are greater or lesser by 10 mg. per cent of blood glucose. This is evidenced by probability values that are greater than .05. The P value that most closely approaches significance is shown between the 80–89 and 90–99 mg. per cent groups. The P value was also checked for all the groups. There were no statistical differences shown except when the 70–79 and 90–99 groups were compared. A P value of <.005 was obtained when the scores of these two groups were analyzed.

B. COMBINATION OF THE TWO ROENTGENOGRAPHIC SIGNS

An analysis of the data will be offered for alveolar bone loss and periodontal widening. The combination of these two signs will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance. There exists a difference in the scoring systems of these two signs, alveolar bone loss being measured on a six point scale and periodontal widening on a three point scale. In order to equate these scores, the periodontal scores were doubled so that a value could be placed on the combination of these two roentgenographic signs.

1. Mean Scores. It appears from Figure 3 that the relationship between fasting blood glucose and the presence of these two signs is parabolic. In other words, alveolar bone loss and periodontal widening are least with fasting blood levels in the 70–79 mg. per cent range. As the fasting blood glucose increases or decreases, the combination of alveolar bone loss and periodontal widening increases.

2. Coefficients of Variation. Table IV shows that the coefficients of variation more or less decrease with an increase in the blood glucose groups. The conclusion to be drawn is that the individual findings more

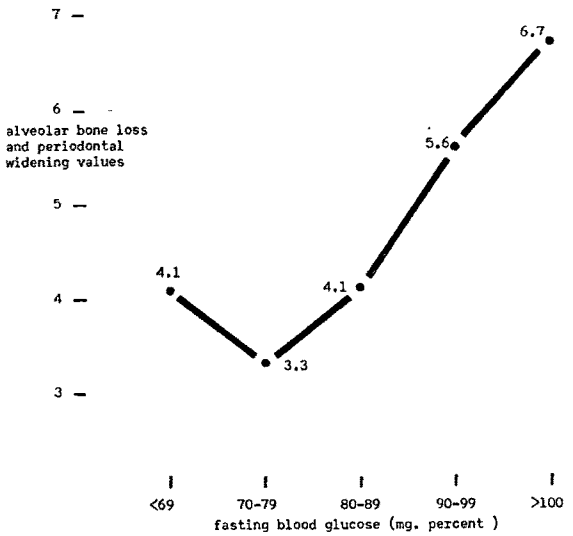


FIG. 3. Relationship of fasting blood glucose to alveolar bone loss and periodontal widening (100 routine dental patients).

TABLE IV

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss and Periodontal Widening Scores	4.1	3.3	4.1	5.6	6.7
Standard Deviation	4.2	2.9	3.8	3.0	2.7
Coefficient of Variation	102	88	93	54	40
Mean Difference	0.8	0.8	1.5	1.1	
Standard Error of Difference	1.30	0.86	1.22	1.49	
Observed t Value	0.615	0.930	1.229	0.738	
Degrees of Freedom	48	69	41	13	
P	.5	>.2	>.2	>.4	

closely cluster about the mean in those cases with hyperglycemia.

3. Statistical Significance. Table IV shows no statistically significant difference between any of the fasting blood glucose groups and their adjacent group or groups. A comparison of each group with all others showed no statistically significant differences except between the 70–79 mg. per cent group, and the 90–99 and >100 mg. per cent groups. The P values here were <.05 and <.01, respectively.

DISCUSSION

The data will be discussed in the following fashion: (1) mean scores, (2) coefficients of variation, and (3) significant probability values. Because of the close correlation between age versus glucose levels and roentgenographic signs versus age, the age factor will be analyzed in terms of blood glucose and roentgenographic signs.

1. *Mean Scores.* An analysis of the mean score values for the two roentgenographic signs, considered independently and in combination, points up the fact that the patterns are parabolic. Moreover, the parabolic picture is more clearcut when the two signs are combined. These data would tend to indicate that there is a range of blood glucose normality for a particular roentgenographic sign or group of signs (Table V) in the general range of 70–79 mg. per cent. This information also suggests that fasting blood glucose levels above and below these narrow ranges are apt to be associated with roentgenographic findings.

2. *Coefficients of Variation.* Generally speaking, the lower the coefficient of varia-

TABLE V

COMPARISON OF MEAN SCORES

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Alveolar Bone Loss	2.4	2.1	2.6	2.9	4.0
Periodontal Widening	0.8	0.6	0.8	1.3	1.2
Alveolar Bone Loss and Periodontal Widening	4.1	3.3	4.1	5.6	6.7

TABLE VI

COMPARISON OF COEFFICIENTS OF VARIATION

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Alveolar Bone Loss	83	71	73	62	28
Periodontal Widening	138	138	125	54	100
Alveolar Bone Loss and Periodontal Widening	102	88	93	54	40

tion, the more the individual scores cluster about the mean. Thus, a coefficient of variation of zero is the ultimate in that it signifies that all subjects provide scores exactly like the mean.

Table VI summarizes the coefficients of variation with respect to the fasting blood glucose groups and the single and combined roentgenographic signs. It can be seen that basically the individual scores cluster more closely in the higher blood glucose groups.

3. *Significant Probability Factors.* Table VII gives a tabulation of the significant probability findings for the two single and combined roentgenographic signs. It is noteworthy that patients having fasting blood glucose levels of >100 mg. per cent show significant differences in the amount of alveolar bone loss when compared to those with levels below 90 mg. per cent. For periodontal widening, the only significant differences occurred when the 70–79 and 90–99 mg. per cent groups were compared. However, significant differences appeared between the 70–79 and 90–99 mg. per cent groups, and the 70–79 and >100 mg. per cent groups, when the two roentgenographic signs were combined. It is clear that the greater the hyperglycemia, the more significant is the difference in these combined roentgenographic signs, when these patients are compared with those having blood glucose levels of 70–79 mg. per cent.

AGE FACTOR

There are some investigators who contend that with increasing age the normal blood glucose values increase. Certainly, the data from which this study is derived indicate that roentgenographic signs, in the main, occur predominantly in the older age groups (Table VIII). Hence, it was thought necessary to analyze the relationship between roentgenographic signs and blood glucose levels in a single age group.

Figure 4 graphically demonstrates the relationship of fasting blood glucose to alveolar bone loss and periodontal widening in the 19 patients between the ages of forty

TABLE VII

COMPARISON OF SIGNIFICANT PROBABILITY VALUES

	Relationship of the Fasting Blood Glucose Groups (mg. per cent)	P Value
Alveolar Bone Loss	<69 with >100	<.050
	70–79 with >100	<.005
	80–89 with >100	<.025
Periodontal Widening	70–79 with 90–99	<.005
Combination of Alveolar Bone Loss and Periodontal Widening	70–79 with 90–99	<.050
	70–79 with >100	<.010

TABLE VIII

PERCENTAGE FREQUENCY OF PATIENTS WITH ROENTGENOGRAPHIC SIGNS BY AGE GROUPS

Age Group	Alveolar Bone Loss	Periodontal Widening	Alveolar Bone Loss and Periodontal Widening
0– 9	0	0	0
10–19	44	22	11
20–29	78	39	39
30–39	96	52	52
40–49	100	74	74
50–59	100	50	50
60–69	100	86	86

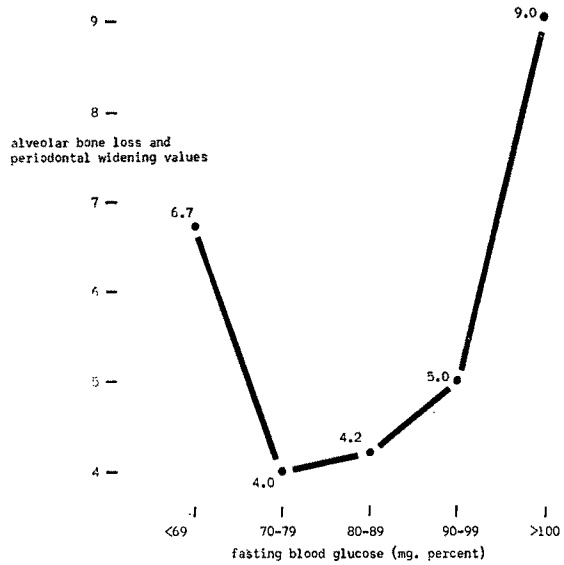


FIG. 4. Relationship of fasting blood glucose to alveolar bone loss and periodontal widening in patients forty to forty-nine years of age.

and forty-nine. An examination of Figure 4 and a comparison between these data and the findings in Figure 3 indicates that the parabola is more exaggerated when the age factor is eliminated. There were no significant differences observed when each group was compared to any other group (Table IX). However, a comparison of Tables IV and IX shows that the individual scores for the one age group cluster more about the mean than was observed for the entire group as judged by the coefficients of variation. Thus it appears that age, or some factor operating with age, may be involved in the relationship of the two roentgenographic signs and glucose metabolism.

SUMMARY

1. This report analyzes the relationship of two roentgenographic signs (*alveolar bone loss* and *periodontal widening*) to the fasting blood true glucose in a group of 100 routine dental patients.

2. An examination of the available data points out the need for evaluating edentulous patients as to the cause of loss of their teeth.

3. An analysis of the mean score values

TABLE IX

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING (40–49 YEAR AGE GROUP)

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss and Periodontal Widening Values	6.7	4.0	4.2	5.0	9.0
Standard Deviation	4.8	1.4	2.0	0.0	4.2
Coefficient of Variation	72	35	48	0	47
Mean Difference		2.7	0.2	0.8	4.0
Standard Error of Difference		2.87	1.06	0.91	3.00
Observed *t* Value		0.94	0.19	0.88	1.33
Degrees of Freedom		8	10	5	2
P		>.4	.5	>.4	>.2

for the two signs, considered independently and when combined, points up the fact that the patterns are generally parabolic. Moreover, the parabolic pattern is more clearcut when the signs are combined than when each sign is analyzed alone.

4. A study of the coefficients of variation shows that, in general, the individual scores cluster more closely about the mean in the higher blood glucose groups.

5. An analysis of the significant probability values shows that the 70–79 mg. per cent fasting blood glucose group has P values of $<.05$ when compared to one of the higher groups for the roentgenographic signs, singly and in combination.

6. The findings in this report suggest that the two roentgenographic signs (alveolar bone loss and periodontal widening) are associated with both hyper- and hypoglycemia.

7. The evidence derived in this report suggests that the age factor, or some other factor operating with age, is associated with hyper- and hypoglycemia in the relationship of these two roentgenographic signs and glucose metabolism.

8. These data suggest that the range of fasting blood glucose normality may be more narrow than is usually accepted.

9. Earlier reports[2,3] have shown that the two-hour blood glucose is a better indicator of glucose metabolism than fasting blood glucose with regard to oral symptoms. A report to follow[7] will analyze the relationship of the two-hour blood glucose to these roentgenographic signs.

E. Cheraskin, M.D.
University of Alabama Medical Center
1919 7th Avenue South
Birmingham 3, Alabama

REFERENCES

1. CHERASKIN, E., and MANSON-HING, L. Normal glucose tolerance pattern: development of blood glucose normality by analysis of oral roentgenographic findings. *Oral Surg., Oral Med. & Oral Path.*, 1960, *13*, 819–835.
2. CHERASKIN, E., and BRUNSON, C. Relationship of fasting blood glucose to oral symptoms. *J. Appl. Nutrition*, 1960, *13*, 2–19.
3. CHERASKIN, E., and FLATLAND, R. Relationship of two-hour blood glucose to oral symptoms. *J. Appl. Nutrition*, 1960, *13*, 66–83.
4. GOLOMB, I. M. Evaluation of relation of diabetes mellitus to periodontal disease. *New York Dent. J.*, 1959, *15*, 525–528.
5. KAPLAN, N. Oral symptomatology of diabetes mellitus. *Pennsylvania Dent. J.*, 1938, *41*, 50–51, 60–61.
6. LOVESTEDT, S. A., and AUSTIN, L. T. Periodontoclasia in diabetes mellitus. *J. Am. Dent. A.*, 1943, *30*, 273–275.
7. MANSON-HING, L., and CHERASKIN, E. Relationship of two-hour blood glucose to roentgenographic signs. *J. Am. Geriat. Soc.*, 1961, *9*, 125–135.
8. MARTINEZ, E. Diabetes in dental practice. *Dent. Abst.*, 1956, *1*, 178.
9. NELSON, N. Photometric adaptation of Somogyi method for determination of glucose. *J. Biol. Chem.*, 1944, *153*, 375–380.
10. PERSON, P. Some aspects of bone metabolism of maxilla and mandible. *New York Dent. J.*, 1957, *23*, 165–170.
11. POLLACK, H., PERSON, P., and KNISHKOWY, E. Diabetes mellitus, its relation to oral pathosis. II. Oral lesions seen in diabetes mellitus. *J. Periodont.*, 1947, *18*, 155–158.
12. RUDY, A., and COHEN, M. M. Oral aspects of diabetes mellitus. *New England J. Med.*, 1938, *219*, 503–508.
13. RUDY, A., and COHEN, M. M. Oral aspects of diabetes mellitus. *J. Am. Dent. A.*, 1942, *29*, 523–534.
14. RUTLEDGE, C. E. Oral and roentgenographic aspects of teeth and jaws of juvenile diabetics. *J. Am. Dent. A.*, 1940, *27*, 1740–1750.
15. SHERIDAN, R. C., JR., CHERASKIN, E., FLYNN, F. H., and HUTTO, A. C. Epidemiology of diabetes mellitus. II. Study of 100 dental patients. *J. Periodont.*, 1959, *30*, 298–323.
16. SOMOGYI, M. Determination of blood sugar. *J. Biol. Chem.*, 1945, *160*, 69–73.
17. SOMOGYI, M. New reagent for determination of sugars. *J. Biol. Chem.*, 1945, *160*, 61–68.
18. STAHL, S. S. Roentgenographic and bacteriologic aspects of periodontal changes in diabetics. *J. Periodont.*, 1948, *19*, 130–132.
19. ZISKIN, D. E., SIEGEL, E. H., and LOUGHLIN, W. C. Diabetes in relation to certain oral and systemic problems; clinical study of caries, tooth eruption, gingival changes, growth phenomena, and related observations in juveniles. *J. Dental Research*, 1944, *23*, 317–331.

BILATERAL BENIGN BONE CYST OF THE OS CALCIS*

By ROBERT O. CHRISTMAN, M.D., † *and* HARVEY P. KOPELL, M.D.‡
NEW YORK, NEW YORK

THE os calcis can be the site of any type of osseous or other connective tissue pathology. As far as can be determined from the literature, the os calcis is neither exempt from nor more prone to any particular lesion. Among lesions described involving the os calcis, benign bone cysts have been sporadically reported.[1–8] In most of the cases mentioned, the cyst was discovered as an incidental finding when the extremity was roentgenographed following trauma. The cysts occurred in the anterior inferior portion of the bone. This is the classic site given in all reports. We would like to describe a case of a benign bone cyst which was found in an unusual location.

REPORT OF A CASE

The patient was a fifty-one year old white female with a congenital kyphoscoliosis who had sustained an injury to her right foot and ankle in a fall. Immediately after the fall there were pain and swelling of the heel and ankle. Weight could not be borne on the affected extremity. There was no history of trauma or disability of the lower extremity previous to the injury. There was no known background of a metabolic or endocrine disturbance. A roentgenogram of the right foot and ankle after admission to the University Hospital revealed a comminuted fracture of the os calcis (Fig. 1). The fracture had occurred at the posterior superior aspect of the bone. The fracture fragments appeared to have composed the posterior superior wall of a cystic lesion.

Because of the unknown nature of the cystic lesion and because of the displaced bone fragments and partial Achilles tendon avulsion, surgical exploration was indicated. At surgery, five days after the trauma, a large unicameral cyst was found. This was roughly ovoid in form with a small, globular outpocketing anteromedially. The cyst contained small blood clots and a moderate amount of serosanguinous fluid. A very thin lining membrane was disclosed through curettage. The specimen submitted to the Pathology Laboratory consisted of fragments of the fractured os calcis with the contents of the curetted cystic area. The bone specimen showed a fairly acellular layer of fibrous tissue lining some parts of the surface of the bony fragments. This fibrous tissue in places revealed evidence of hemorrhage. The curetted specimen consisted of dense fibrous tissue and clot. The pathologic diagnosis was "fragments of bone cyst."

The laboratory tests included a complete blood count, urinalysis and sedimentation rate, serum calcium, phosphorus, phosphatase (acid and alkaline), total proteins, A-G ratio and creatinine, urinary creatinine, calcium and phosphorus, and tests for tubular resorption of phosphorus. The findings were all within normal limits.

Figure 2 shows the right ankle after surgical exploration and removal of the comminuted fragments. For purposes of comparison, a roentgenographic study of the left os calcis was done (Fig. 3 and 4). Surprisingly, this also revealed a triangular area of radiolucency at the posterior superior portion of the os calcis. There was an apparent horizontal septal division inferiorly. The lesion was marginated by a line of increased bone density. Bone trabeculation and density were normal throughout the remainder of the os calcis and visualized extremity. We assumed this to represent a benign cyst of the opposite os calcis. A skeletal survey was performed to rule out other cystic lesions. None were found. The patient's postoperative period was uneventful.

DISCUSSION

A major problem confronting the roentgenologist on the demonstration of a bone lesion is to decide whether it is benign or malignant. The expression of this potential

* From the Departments of Radiology and Orthopedic Surgery of the New York University School of Medicine and the Bellevue Hospital Center.
† Assistant Professor of Radiology, New York University School of Medicine; Associate Attending Roentgenologist, University Hospital; Assistant Visiting Roentgenologist, Bellevue Hospital.
‡ Associate Professor of Orthopedic Surgery, New York University Post-Graduate Medical School; Associate Visiting Orthopedic Surgeon, Bellevue Hospital; Assistant Attending Orthopedic Surgeon, University Hospital.



Fig. 1. Roentgenogram of the right ankle showing comminuted fragments, displaced superiorly, which appear to have composed the posterior superior wall of a cystic lesion.



Fig. 2. The right ankle after surgical exploration and removal of the comminuted fragments.



Fig. 3. Roentgenogram of the left ankle demonstrating a radiolucent lesion with a densely marginated border in the posterior superior portion of the os calcis.



Fig. 4. Roentgenogram of the left os calcis in a sagittal view.

is the same for the os calcis as for other areas of the osseous system. A sharply delineated lesion with a discrete condensed border points to the benign nature of the abnormality. It is possible to have a sharply delineated lesion in a malignant process, as in a myeloma. It is also possible to have a sharply delineated lesion in a benign process such as gout or an eosinophilic granuloma. However, the presence of a densely marginated border almost always indicates a benign entity. When such a lesion which does not demonstrate significant expansion from within is presented, it reinforces the impression of benignity. Laboratory data are important in confirming the roentgenographic impression.

We believe that cysts of the os calcis are probably of greater frequency than the relatively few reports would lead one to suppose. The available literature does not mention the possibility of a contralateral lesion. The fact that the lesion in this case is presumed to be bilateral (the contralateral side was not biopsied) and that it is not in the accepted classic location does not detract from the diagnosis of a benign bone cyst.

Benign bone cysts can be found in any part of the skeletal system derived from endochondral bone. These lesions occur near an epiphyseal line and by juxtaepiphyseal growth migrate toward the diaphysis. The basic formation of the cyst comes about through a limited disorder of an early member of the osteogenic sequence rather than through disturbance of a mature bone element. We used the term "limited" above because, once the cyst is formed, normal bone formation and growth may occur at the site from which the cyst arose. This explains the normal bone intervening between the cyst and the epiphyseal line. The relatively dense surrounding border may represent a proliferative response to the lining membrane; it may result from the pressure of the enclosed fluid or from diffusion of osteogenic inductive elements from the fluid or membrane. If this border were purely a mechanically induced phenomenon caused by diversion of stress lines, one would expect, in an eccentrically placed lesion in a long bone, marked asymmetry of the margin; where the border parallels the main force lines, it should be thicker. In general, the bone density surrounding the cyst bears no relationship to the axial stress configuration, so the border apparently is not simply a mechanically induced phenomenon.

SUMMARY

Bone cysts of the os calcis have been described in the literature. Previous authors have stressed the localization of a benign cyst to the anterior inferior portion of the os calcis. A case has been reported in which the lesion occurred at the posterior superior portion of the os calcis with the roentgenographic demonstration of a similar lesion in the contralateral bone.

Robert O. Christman, M.D.
University Hospital
303 East 20th Street
New York 3, New York

REFERENCES

1. Copleman, B., Vidoli, M. F., and Crimmings, F. J. Solitary cyst of calcaneus. *Radiology*, 1946, *47*, 142–148.
2. Hundley, J. M. Solitary bone cyst of os calcis (form of osteitis fibrosa). *J. Arkansas M. Soc.*, 1948, *45*, 7–8.
3. Jaffe, H. L. Tumors and Tumorous Conditions of the Bones and Joints. Lea & Febiger, Philadelphia, 1958, chapter 5.
4. Janes, J. M. Localized bone cyst of os calcis. *J. Bone & Joint Surg.*, 1946, *28*, 182.
5. Miles, J. E., and Degenshein, G. A. Solitary bone cysts. *Am. J. Surg.*, 1956, *91*, 170–177.
6. Schinz, H. R., Baensch, W. E., Friedl, E., and Uehlinger, E. Roentgen-Diagnostics. First American edition. Volume II. Grune & Stratton, Inc., New York, 1951-1952, pp. 873–876.
7. Schlüter, K. Ossifikationsanomalien im Kalkaneus, zugleich ein Beitrag zum Auftreten von Ossifikationszentren im Tarsus. *Ztschr. Orthop.*, 1955, *87*, 37–55.
8. Verstandig, C. C. Solitary unicameral cyst of os calcis. *New England J. Med.*, 1947, *237*, 21–22.

Vol. 86, No. 2

THE ROENTGENOLOGIC FEATURES OF ACROMEGALY

By ERICH K. LANG, M.D.,* *and* WALTER T. BESSLER, M.D.
BALTIMORE, MARYLAND

THE advanced stages of acromegaly show characteristic roentgenologic changes of the bones and joints. Particularly, the skull roentgenograms may prove to be of diagnostic importance. In most cases, there is enlargement and erosion of the sella caused by the increasing pressure of the hyperplastic pituitary gland. The roentgenologic manifestations in other structures occur rather late in the course of the disease; nevertheless, these changes are of great importance for the understanding of the dynamics and pathogenesis of this disease.

PATHOGENESIS

It is well established that the disease is due to an increase in function and size of a portion of the anterior lobe of the pituitary gland.[12] Pathologically, the lesion is most commonly an eosinophilic adenoma but, occasionally, mere hyperplasia of the eosinophilic cells without actual adenoma formation or even eosinophilic adenocarcinoma or chromophobe adenoma may give rise to this clinical syndrome. The expansile growth of the pituitary neoplasm causes the familiar erosion of the osseous wall of the sella and the characteristic bulging of the diaphragm. The absence of roentgenologic changes of the sella, however, does not allow one to exclude the diagnosis of acromegaly, since the pituitary gland may show marked increased hormonal activity without significant enlargement and hyperplasia. The most obvious manifestations of the disease are caused by the liberation of excessive amounts of growth hormone. The mere pressure of the tumor, however, may result in a disturbance of the other pituitary functions of the uninvolved portion of the gland or may cause symptoms by pressure on adjacent nerves.[4]

CLINICAL AND ROENTGEN FEATURES

The excessive amount of growth hormone or somatropin has a direct effect on the osseous, cartilaginous and connective tissues.[9,10,11,21] In childhood, the overproduction of growth hormones causes so-called pituitary giantism. If the onset of the disease is during early adolescence, giantism with acromegalic features is the usual form of presentation. If, however, the onset of the disease is in adulthood, one observes the typical acromegalic form.[1,2,3]

The accentuation of normal appositional growth of bone is the most significant roentgenologic feature of this disease. Marked spur formation and relatively early appearance of degenerative osteoarthritic changes are noted in the majority of patients.[7,8] This appositional bone growth is most characteristically demonstrated in areas of normal bone prominence such as tuberosities and sites of muscular or ligamentous attachment.[8] Periosteal new bone formation, particularly along the anterior margins of the vertebral bodies, is one other classic manifestation of this type of process.[6,10,21] The typical facial appearance of the patient is caused by the prominence of the zygomatic arches and supraorbital ridges, as well as by prognathism. These changes again are due to appositional bone growth as well as overgrowth and elongation of the mandible and lower teeth.

Elongation of the bones by endochondral growth is seen more rarely than appositional growth, mainly because most patients are observed during puberty or later, when most epiphyses have fused. It is stated, however, that even in this age group there may be limited endochondral growth at special sites.[18,24] Weinberg points out that, particularly in the deep layers of the articular cartilage, there may be endo-

* Department of Radiology, The Johns Hopkins Hospital, Baltimore, Maryland.

chondral growth resulting in increasing length of the tubular bones, even after what appears to be complete obliteration of the epiphyseal cartilage. Marked cartilage proliferation, however, is seen in the regions of the costochondral junctions, as well as in the joints of the hands and feet.[18,23] The costochondral junction of the ribs shows marked proliferation below both the perichondrium and the periosteum. This results in marked prominence of this region, thereby causing the so-called acromegalic rosary. A marked proliferation of joint cartilage has long been recognized as a classic feature of this disease.[7,9,20,23] The nature of the rapidly formed cartilage, however, appears to be in considerable doubt. Early degenerative changes are found in periarticular areas, and particularly so in the region of the junction of the perichondrium and periosteum.

The collagen tissue also shows a marked response to the excessive amount of growth hormone. Thickening of the skin, increase of the periportal connective tissues, increase of the supporting network of the bronchi, changes in the connective tissues supporting the renal parenchyma, and even interstitial tissue hypertrophy of the pancreas have been reported.[8,14,16] There may be painful swelling of the finger joints and generalized enlargement of the soft tissues of the fingers, as well as actual thickening of the synovia as seen on microscopic sections.

Generalized and localized osteoporosis is a well-known feature of this disease.[7,18,19,22] The most typical example of localized resorption of bone is the classic enlargement of the frontal sinuses. This is undoubtedly caused by a dual process, *i.e.*, both bone resorption and apposition, resulting in a large frontal sinus with bulging of the supraorbital ridges. The tubular bones are consistently subjected to both resorption and apposition. Curschmann[7] and Kellgren *et al.*[18] have emphasized the excessive modeling of tubular bones resulting in abnormally thin, spindly phalanges.[7,18]

ROENTGEN FINDINGS

The material analyzed for this paper consists of 31 patients, most of them with long-standing symptoms (mean duration of disease, nine years). The age distribution ranged between twenty-four and seventy-seven years. The roentgen findings are tabulated in Tables I and II.

SKULL

The classic changes of the sella were encountered in 29 of 31 patients (Table I). Only 2 patients showed a sella which was considered normal in size and contour. The most frequently encountered manifestation was generalized enlargement of the sella with rarefaction of the dorsum and associated demineralization of the anterior and posterior clinoids. Complete destruction of the dorsum of the sella was encountered in only 4 patients and did not necessarily correlate with a particularly active form of the disease (Fig. 1, *A* and *B*). The fact that the disease was chronic in the patients examined in this series may well account for the high percentage of positive findings in the sellar region. Steinbach *et al.*[21] also noted a significantly large number of positive findings of sellar destruction (81 per cent).

The second most common feature was prominence and enlargement of the frontal sinuses; this was present in 23 of 31 patients (Table II). Contrary to the observation of Steinbach *et al.*[21] there was no sig-

TABLE I

OSSEOUS CHANGES OF THE SELLA TURCICA

Changes	No. of Cases
Complete destruction of the dorsum of the sella	4
Generalized enlargement of the sella with rarefaction of the dorsum	19
Slight enlargement of the anteroposterior diameter of the sella	6
No apparent change	2
Total	31

TABLE II

ROENTGEN FINDINGS OF ACROMEGALY

Site and Changes	Appositional growth of bone, thickening and osseous overgrowth	Osseous resorption and bone modeling	No abnormal findings
	No. of Cases	No. of Cases	No. of Cases
Skull (31 Cases)			
Cranial vault—diffuse manifestations	11	4	7
Cranial vault—circumscribed manifestations	7	2	
Occipital spur formation	12		19
Prominence and enlargement of sinuses			
Frontal	23		8
Maxillary	21		10
Mastoid enlargement	13		18
Hands (23 Cases)			
Heads and bases of phalanges	6		17
Phalanges and metacarpal shafts	6	3	14
Phalanges and metacarpal shaft elongations (endochondral ossification)	2		21
Joint cartilage (widening of joint spaces)	14		9
Osteoarthritis	11		12
Exostoses	9		14
Large tufts of terminal phalanges	11		12
Diffuse osteoporosis		7	16
Feet (13 Cases)			
Shafts of phalanges		11	2
Heads and bases of phalanges and metatarsals	10		3
Tufts of distal phalanges	8		5
Tarsals	3	6	4
Osteoarthritis	8		5
Joint cartilage hypertrophy (widening of the joint space)	5		8
Thoracic Spine and Thoracic Cage (20 Cases)			
Length of vertebral bodies (particularly anterior circumference apposition)	10	5	5
Intervertebral disks	12	10*	
Calcified disks	4		16
Osteoarthritis	15		5
Increased anteroposterior diameter of the chest	12		8
Anterior bulging of the sternum	16		4
Lumbar Spine (6 Cases)			
Intervertebral disks	6		
Length of vertebral bodies (modeling of posterior circumference of bodies)		4	2

* Some cases showed both resorption and bone growth at different levels.

nificant distribution of the male and female patients in this respect. Two-thirds of the entire group also showed correspondingly large maxillary antra. An evaluation of the degree of pneumatization of the periantral air cells was quite difficult. However, it was felt that about one-third showed probably increased pneumatization. The mastoid tip in particular often showed extensive pneumatization and prominence. About one-third of all patients, most of them male, showed marked prominence of the occipital

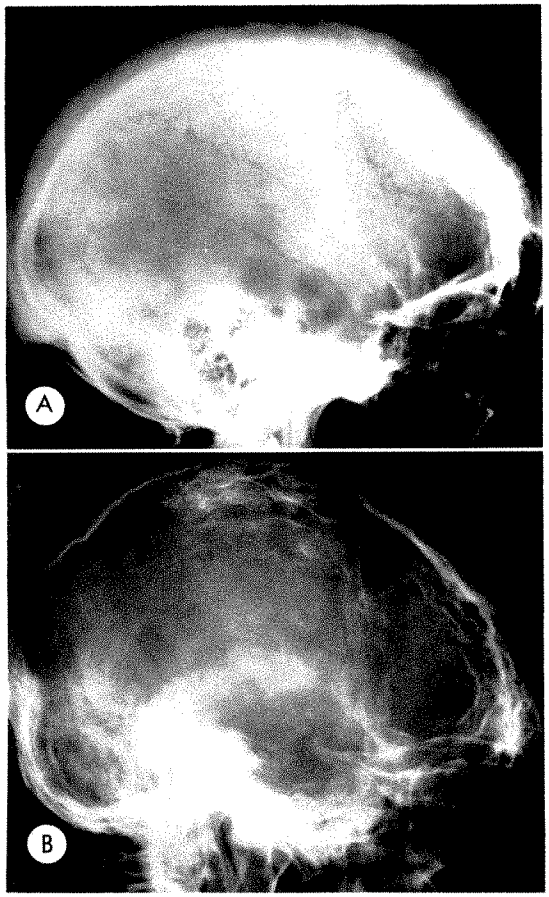


FIG. 1. (*A*) Lateral roentgenogram of the skull showing complete destruction of the dorsum of the sella and posterior clinoids with marked thickening of the entire calvarium and hyperostosis frontalis interna. The occipital protuberance is quite prominent, forming a spur-like hyperostosis at the point of insertion of the nuchal ligament. (*B*) There is almost complete destruction of the dorsum of the sella and the posterior clinoid processes. Note the selective thinning of the parietal bones.

protuberance (Fig. 1*A*). The occipital protuberance projected in the form of a spur apparently formed by marked bone apposition at the point of the insertion of the nuchal ligament.

One of the most interesting observations which has been made in the literature on acromegaly is the presence of both thinning and thickening of the cranial vault.[13,18] This either involves the entire cranial vault or is localized to the frontal and parietal bones. In our series, thickening of the tables appeared to be much more common than thinning. Localized thickening presented most often as hyperostosis frontalis interna. Localized thinning was most commonly seen in the region of the parietal bone (Fig. 1*B*). Both appositional and resorptive changes resulted in the loss of the typical differentiation between the external and internal tables and the diploë. The diploic space exhibited a marked increase in density and a coarsening of its trabecular pattern. A gradual obliteration of the entire diploic space takes place as the disease progresses. There was often associated soft tissue proliferation, probably secondary to thickening of the galea aponeurotica. This proliferation, however, appeared to be independent of osseous hypertrophy. Prominence of the supraorbital ridges and zygomatic arches was a salient feature. The enlargement and elongation of the mandible was difficult to evaluate from our series because most of the roentgenograms failed to show the entire mandible and, hence, did not allow proper interpretation of this region.

HANDS

The first classic description of the so-called "spade hand" dates from 1886.[19] These changes are predominantly caused by soft tissue hypertrophy. Eighteen of 23 patients examined in this series showed marked thickening of the soft tissues of the fingers (Fig. 3). The typical squaring and thickening of the phalanges and metacarpals, however, was only seen in one-fourth of all patients. Three of 23 patients exhibited narrowing of the shafts of the phalanges. This phenomenon was first described by Marie[19] and appears to be limited to juvenile patients. The apparent narrowing is probably caused by a combination of accentuated growth in length produced by excessive endochondral activity and abnormal resorption of bone along the shaft.[7]

The most characteristic changes in the hands are the abnormally wide joint spaces, undoubtedly caused by marked hypertrophy of cartilage (14 of 23 cases.)[3] De-

generative osteoarthritic manifestations were also present and are commonly seen in both younger and older age groups affected with this disease. Small exostoses occur in the areas of tendon or ligament insertion. These changes are best seen in the heads of the metacarpal bones. The enlargement of the ungual tufts of the terminal phalanges is one of the salient features more commonly seen in males. Some patients may show a combination of hypertrophic changes of the distal phalanges associated with osteoporosis, thinning and resorptive processes of the proximal phalanges and metacarpals. This combined type, however, was seen in only 2 patients and appeared to be quite rare.

FEET

The changes of the osseous structures of the feet are similar to those of the hands, combined hypertrophic and resorptive changes being very frequent. Nearly all of our cases showed characteristic narrowing of the shafts of the phalanges contrasting sharply with the disproportionately broad and square bases and heads. At the same time, there was usually thickening and a square appearance to the shafts of the metatarsals (Fig. 4). These changes probably best attest to the dynamics of the disease, showing marked resorptive changes along the shafts of the phalanges as well as apposition and new bone deposition in other areas of the same osseous structures.

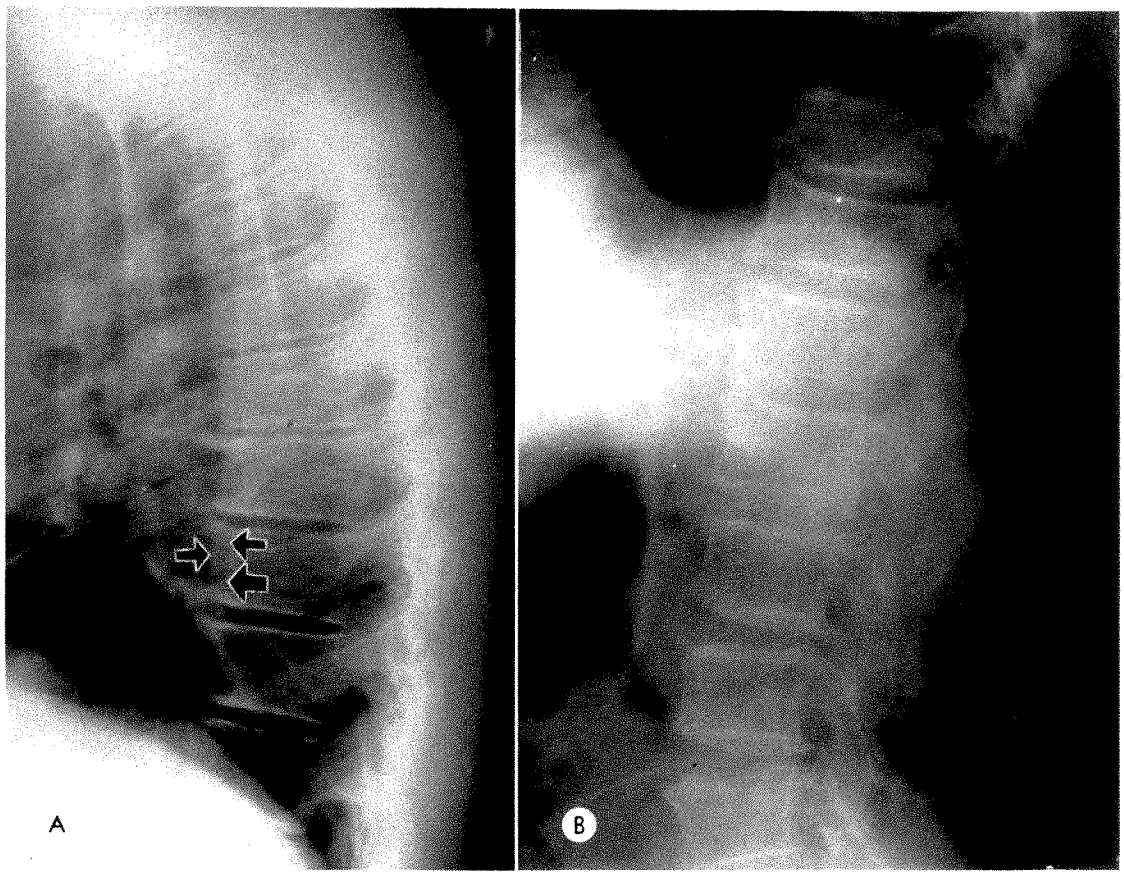


FIG. 2. (*A*) Lateral roentgenogram of the dorsal spine demonstrating the increased length of the dorsal vertebral bodies caused by appositional new bone formation along the anterior aspects. The old anterior margin can still be seen as a faint cortical shadow (arrows). (*B*) Note the marked concavity of the posterior aspect of the lumbar vertebrae caused by bone resorption. There is marked widening of the lumbar disks and also some osteoarthritic changes.



Fig. 3. Marked hypertrophy of the ungual tufts, widening of the metacarpophalangeal joint spaces (secondary to cartilage hypertrophy), and spur and exostosis formation at points of tendon insertion are the salient features. Note the marked thickening of the soft tissues.

In our series, some of the metatarsals also showed thinning, which is considered to be a rather rare finding. Polyarthrosis of both phalanges and metatarsals was commonly demonstrated. Hypertrophy of the joint cartilage was less commonly seen in the lower extremities (5 of 13 cases).

SPINE

The first classic description of the changes of the spine was published by the Austrian pathologist, Erdheim.[10] Since then, many authors have re-emphasized the importance of the accentuation of the physiologic kyphosis of the upper dorsal spine and the compensatory hyperlordosis of the lumbar spine in the diagnosis of this disease.[5,6,7,10,13,18,21,23] The most distinctive changes, however, are undoubtedly those caused by the selective apposition and resorption of bone along the dorsal and lumbar spine.[21] In this series, there appeared to be a significant difference in the changes of the dorsal and lumbar vertebrae. The dorsal vertebrae predominantly showed apposition, particularly along the anterior aspect of the vertebral bodies (Fig. 2*A*). These changes appear to be characteristic of the disease and deserve special attention when reading lateral chest roentgenograms. Conversely, the lumbar spine predominantly showed modeling of the posterior aspects of the vertebral bodies, undoubtedly secondary to resorptive processes (Fig. 2*B*). Both lumbar and dorsal spines may show increased height of the intervertebral disk spaces. In a few instances, however, there was also evidence of degeneration and secondary narrowing of some of the disk interspaces. This was only seen



Fig. 4. Posteroanterior roentgenogram of the foot. Exaggerated overmodeling of the shafts of the phalanges contrasts with the hypertrophic changes of the heads and bases. Septa can be discerned in the soft tissues, indicative of edema.

in the region of the lower dorsal spine. The upper dorsal spine and the lumbar spine showed a uniform increase in the intervertebral disk spaces. Osteoarthritic changes were observed in 15 of 20 cases. The spurs seen in these patients, however, are much more uneven and irregular than those commonly associated with degenerative osteoarthritic disease in older age groups. Furthermore, there appeared to be a significant absence of sclerosis of the end plates of the vertebral bodies, which is characteristic of nonacromegalic degenerative osteoarthritis. These changes are believed to differentiate the osteoarthritis seen in acromegaly from that seen in the old age groups.

THORACIC CAGE

The anteroposterior diameter of the chest is often increased in acromegalic patients; this is due to the more or less horizontal position of the manubrium and the associated ventral displacement of the sternum. The ventral ends of the ribs are often thickened and the costochondral cartilage junction may show knotty proliferations characteristic of the acromegalic rosary. Finlay and MacDonald[13] first commented on the fusiform widening of the costochondral junctions, which was present in 50 per cent of their cases. Waine *et al.*[23] felt that the cartilage of the costochondral junction was surrounded and enveloped in a sheath of new bone formed by the proliferating periosteum. Resorptive processes along the ribs are quite rare and are usually localized to only a few segments.

HIP JOINTS

In addition to the tabulated changes (Table II), one may frequently see degenerative changes of the hip joints. It is postulated that these changes are caused by a qualitatively inferior cartilage which degenerates early and hence exposes the osseous components of the joints to friction. In these areas very severe reactive sclerosis may be encountered and there may in fact be partial destruction of the osseous components. Genuine degenerative osteoarthritic spur formation is then seen.

DISCUSSION

The diagnosis of acromegaly remains primarily a clinical problem. Roentgenologic studies, however, offer a method for objective comparison of the progression or regression of changes caused by this disease. One of the aims of careful follow-up is to determine whether the disease is quiescent or active and what effect therapy has had.

Three of our patients have been followed for a number of years with multiple roentgenographic studies both prior to and after definitive therapy. These patients showed progressive osseous and soft tissue changes before therapy. The regression of these changes after radiation therapy or surgical extirpation of the tumor was not always dramatic. Initially there was a marked reduction of the soft tissue swelling of the fingers. This observation has been confirmed by many authors but attempts to quantitatively assess this finding have thus far failed.[8,15,17] The regression of soft tissue swelling was only observed in the first few weeks and failed to reach complete restoration to normal, thus leaving residual enlargement which was probably due to irreversible connective tissue hyperplasia. Neurologic and ophthalmologic defects also showed excellent early regression, since these changes had been caused by pressure of the tumor mass which responded rapidly to therapy. Changes of the osseous system appeared to show little effect from the changed hormonal levels. In fact, with the exception of recalcification of the clinoids and sella, there was hardly any regression of the existing osseous changes. Only 1 patient showed definite bone apposition along the shafts of the tarsal phalanges which had demonstrated the classic modeling and thinning during the active form of the disease. These changes became apparent one and one-half years after a tumor dose of

3,500 r had been delivered via five external ports to the sellar region.

Pantopaque ventriculography, exactly outlining the floor of the third ventricle, will aid greatly in the evaluation of the response of suprasellar tumor masses treated with external radiation therapy or implantation of radioactive materials.

SUMMARY

Bone apposition along the anterior aspect of the vertebral bodies of the dorsal spine and the increased height of the intervertebral disks are considered to be characteristic roentgenologic features of acromegaly. The absence of sclerosis of the end plates of the vertebral bodies offers a criterion for differential diagnosis between degenerative and acromegalic osteoarthritis of the spine. A combination of bone apposition and resorption, characteristic of this disease, is particularly frequent in the foot.

The high incidence of sellar changes is felt to reflect the long mean duration of the disease in this series.

Soft tissue swelling as well as neurologic and ophthalmologic changes caused by pressure of the tumor mass show a marked and early response to treatment. The osseous changes are, in general, irreversible.

Erich K. Lang, M.D.
Department of Radiology
Johns Hopkins Hospital
Baltimore 5, Maryland

REFERENCES

1. ARNOLD, J. Weitere Beiträge zur Akromegaliefrage. *Arch. f. path. Anat.*, 1894, *135*, 1–78.
2. ATKINSON, F. R. B. Acromegaly. John Bale Sons & Danielsson, London, 1932.
3. ATKINSON, F. R. B. Acromegaly from study of literature, 1931–1934. *Endokrinologie*, 1936, *17*, 308–320.
4. BAILEY, P. Intracranial Tumors. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1948.
5. BOHATIRCHUK, F. Ageing vertebral column (macro- and historadiographical study). *Brit. J. Radiol.*, 1955, *28*, 389–404.
6. CHESTER, W., and CHESTER, E. M. Vertebral column in acromegaly. AM. J. ROENTGENOL. & RAD. THERAPY, 1940, *44*, 552–557.
7. CURSCHMANN, H. Über regressive Knochenveränderungen bei Akromegalie. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1905–1906, *9*, 83–92.
8. CUSHING, H., and DAVIDOFF, L. M. Pathological findings in four autopsied cases of acromegaly, with discussion of their significance. New York, Rockefeller Institute for Medical Research, Monograph No. 22, 1927.
9. ERDHEIM, J. Die Lebensvorgänge im normalen Knorpel und seine Wucherung bei Akromegalie. Springer, Berlin, 1931.
10. ERDHEIM, J. Über Wirbelsäulenveränderungen bei Akromegalie. *Virchows Arch. f. path. Anat.*, 1931, *281*, 197–296.
11. ERDHEIM, J. Die pathologisch-anatomischen Grundlagen der hypophysären Skelettveränderungen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1935, *52*, 234–245.
12. EVANS, H. M., and LONG, J. A. Characteristic effects upon growth, oestrous, and ovulation induced by intraperitoneal administration of fresh anterior hypophyseal substance. *Anat. Rec.*, 1922, *23*, 19–54.
13. FINLAY, J. M., and MACDONALD, R. I. Acromegaly. *Canad. M.A.J.*, 1954, *71*, 345–352.
14. GERSHBERG, H., HEINEMANN, H. O., and STUMPF, H. H. Renal function studies and autopsy report in patient with gigantism and acromegaly. *J. Clin. Endocrinol.*, 1957, *17*, 377–385.
15. HAMBERGER, C. A., HAMMER, G., NORLEN, G., and SJOGREN, B. Hypophysectomy in acromegaly. *J. Clin. Endocrinol.*, 1959, *19*, 1500–1502.
16. HEJTMANCIK, M. R., BRADFIELD, J. Y., JR., and HERRMANN, G. R. Acromegaly and heart. *Ann. Int. Med.*, 1951, *34*, 1445–1456.
17. HURXTHAL, L. M., HARE, H. F., HORRAX, G., and POPPEN, J. L. Treatment of acromegaly. *J. Clin. Endocrinol.*, 1949, *9*, 126–148.
18. KELLGREN, J. H., BALL, J., and TUTTON, G. K. Articular and other limb changes in acromegaly; clinical and pathological study of 25 cases. *Quart. J. Med.*, 1952, *21*, 405–424.
19. MARIE, P. Sur deux cas d'acromégalie. *Rev. d. méd.*, 1886, *6*, 297–333.
20. REINHARDT, W. O., and LI, C. H. Experimental production of arthritis in rats by hypophyseal growth hormone. *Science*, 1953, *117*, 295–297.
21. STEINBACH, H. L., FELDMAN, R., and GOLDBERG, M. B. Acromegaly. *Radiology*, 1959, *72*, 535–549.
22. VON RECKLINGHAUSEN, F. Über die Akromegalie. *Arch. f. path. Anat.*, 1890, *119*, 36–53.
23. WAINE, H., BENNETT, G. A., and BAUER, W. Joint disease associated with acromegaly. *Am. J. M. Sci.*, 1945, *209*, 671–687.
24. WILLIAMS, R. H., Editor. Textbook of Endocrinology. Second edition. W. B. Saunders Company, Philadelphia, 1955.

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PRIMARY HYPERPARATHYROIDISM: A CORRELATION OF ROENTGEN, CLINICAL, AND PATHOLOGIC FEATURES*

By HOWARD L. STEINBACH, M.D.,[1] GILBERT S. GORDAN, M.D.,[2] EUGENE EISENBERG, M.D.,[2] JACKSON T. CRANE, M.D.,[3] SOL SILVERMAN, D.D.S.,[4] *and* LEON GOLDMAN, M.D.[5]
SAN FRANCISCO, CALIFORNIA

THAT the parathyroid glands are involved in the regulation of the calcium stores of the body and in maintenance of the mineralization of the skeleton has been known for many years.[1,16,18,19,24] The roentgen appearance of grossly abnormal bone produced by excessive secretion of the parathyroid hormone has also been adequately described.[6,21,22] The problem of determining the earliest roentgen manifestations of hyperparathyroidism, however, has not been extensively investigated, and this study will attempt to do so.

PHYSIOLOGY

Phosphate homeostasis is maintained by the action of the parathyroid hormone on the renal tubular transport of phosphate. The parathyroid hormone also maintains the blood calcium level by mobilization of calcium from bone mineral. An excess of the hormone, therefore, causes hypercalcemia and hyperphosphaturia.[7,12] If phosphate loss is not replaced by dietary phosphate intake, hypophosphatemia results.

Evidences of excessive parathyroid activity result either from primary parathyroid overfunction, such as in adenoma or primary hyperplasia, or from secondary hyperfunction as in steatorrhea or renal failure. The initiating event for secondary hyperparathyroidism is probably hypocalcemia; whether hyperphosphatemia has a similar effect is controversial. The kidney is also affected by hyperparathyroidism. Initially, functional impairment consisting of inability to concentrate urine and to conserve phosphate is seen. Later, calcium salts are found in the cells and lumen of the convoluted tubules. Still later, calcification and fibrosis of the parenchyma progress to renal insufficiency.[15] Secondary pyelonephritis and parenchymal calcification further complicate the roentgenologic appearance of calcium deposition.

Much has appeared in the literature on metastatic calcification of the soft tissues. This calcification is of two types: (1) that due to hypercalcemia, as in calcific conjunctivitis, and (2) that resulting from a combination of hypercalcemia and the hyperphosphatemia of renal failure, as with keratopathy and calcification of the alveoli of the lungs, vessel walls, gastric mucosa, cartilaginous and periarticular structures, and other tissues. Curiously, metastatic calcification of soft tissues occurs concomitantly with decalcification of the skeleton.

Hyperparathyroidism may cause disturbances of the urinary tract and skeletal system; it may give rise to symptoms of resultant acute hypercalcemia, or it may be present without evident symptoms. The urinary tract symptoms consist of hyposthenuria, polyuria, nocturia, polydipsia, and dehydration, most commonly observed in patients with severe hypercalcemia. The most common urinary tract complications are nephrolithiasis, pyelonephritis, and nephrocalcinosis. Skeletal involvement may first be manifested by fractures with little or no trauma, but rarely by bone pain and tenderness. Localized expansion, bending,

* From the University of California, School of Medicine, San Francisco, California. Aided by a grant from the United States Public Health Service, Grant A-2589 GM.
[1] Department of Radiology.
[2] Department of Medicine.
[3] Department of Pathology.
[4] Department of Oral Medicine.
[5] Department of Surgery.

TABLE I

GROSS PATHOLOGY IN 112 CASES OF PROVED HYPERPARATHYROIDISM*

Single adenoma	92
Multiple adenomas	10
Primary (wasserhelle) hyperplasia	9
Carcinoma with metastasis	1

* From: Black, B. M.[4] Hyperparathyroidism. Charles C Thomas, Springfield, 1953, p. 20.

or fracture may cause bone deformities.

The clinical symptoms of acute hypercalcemia are anorexia, nausea, vomiting, constipation, abdominal distention, thirst, nocturia, polyuria, weakness, and, when the serum calcium is exceedingly high, lethargy, somnolence, or psychosis. Peptic ulcer is extremely common in patients with hyperparathyroidism. Hellström[15] reported that 33 per cent of the men and 5 per cent of the women in his series had peptic ulcers. Many more have ulcer-like symptoms, approximately 45 per cent of all patients with hyperparathyroidism presenting themselves because of gastrointestinal disturbances.

The keystone of diagnosis is hypercalcemia, often of mild degree, requiring unusually accurate laboratory procedures for recognition and evaluation. Hypophosphatemia, formerly the rule, is now found in only about 40 per cent of cases. Hyperphosphaturia, demonstrated by a low rate of tubular reabsorption of phosphate, is uniformly present. Similar chemical abnormalities occur in other disorders, such as myeloma, sarcoidosis, and neoplasms.

PATHOLOGY

Primary hyperparathyroidism may be caused by a single adenoma, multiple adenomas, primary hypertrophy and hyperplasia, chief cell hyperplasia, or carcinoma.[4,8] The incidence at the Mayo Clinic of these lesions as causes of hyperparathyroidism is shown in Table I. Chief cell hyperplasia may be associated with islet cell adenomas of the pancreas, chromophobe adenomas of the pituitary, and adrenal cortical hyperplasia.[8]

Osteitis fibrosa cystica generalisata affects the entire skeleton. The earliest and most severe lesions appear in regions where bone production and the invariably associated bone destruction or remodelling occur most rapidly.[16] In children, some of the most pronounced changes are in the metaphyses adjoining the growth plate where calcified cartilage is replaced by primitive bone which, in turn, undergoes remodelling through the mechanism of osteoclastic resorption and surface accretion[16] (Fig. 1). In both adult and child, early lesions usually appear along the periosteal surface of the cortex, particularly at the point of tendinous or ligamentous attachments. Sites of injury, mechanical pressure, or tension, with osteoclastic bone destruction and osteoblastic repair, appear to be more sensitive to the parathyroid hormone. The reaction is the opposite of that in acro-

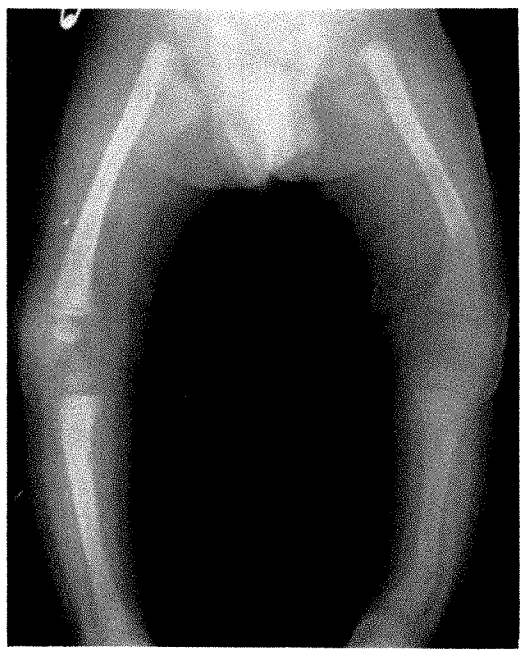


FIG. 1. Hyperparathyroidism resulting from hyperplasia in a ten week old child. A diffuse demineralization and bending deformities of the distal ends of the femora, tibiae, and fibulae are seen. The most marked bone destruction is in the distal femur where a horizontal linear radiolucent shadow is visible proximal to the zone of provisional calcification. (Courtesy, Doctor G. Melvin Stevens, Palo Alto Medical Clinic.)

TABLE II

AGE AND SEX OF PATIENTS WITH BONE INVOLVEMENT STUDIED ROENTGENOGRAPHICALLY

	No. of Cases	Males	Females	Average Age (yr.)
Total number of patients	47	24	23	45 (Range 17–70)
Patients with bone involvement	13 (28%)	7	6	42 (Range 17–64)

megaly in which bone production is greatest at points of stress. Weinmann and Sicher[24] found characteristic histologic lesions of osteitis fibrosa cystica in bone after the extraction of an infected tooth, and adjacent to inflammatory polyps in the maxillary sinus of patients with moderate renal insufficiency and secondary hyperparathyroidism.

The mechanism by which the parathyroid hormone causes bone to be reabsorbed is not fully understood, but probably the resorption is effected by osteoclasts altering the bony matrix and minerals which become soluble and pass into the tissue fluids. This dissolution of bone substance involves the stable, fully mineralized bone, most of which is not accessible to the rapidly acting transfer mechanism. The bone reabsorbs only along the surfaces of the finer structures.[18,19,24]

The well-developed histologic lesion is caused by osteoclastic resorption of bone, associated with reparative fibrosis and formation of poorly calcified immature new bone. The compact, cortical bone is replaced by spongy bone. Localized accumulations of fibrous tissue and giant cells, called "brown tumors," may produce expansion of bone similar to giant cell and other benign tumors. "Brown tumors" undergoing necrosis and liquefaction produce cysts.[16]

ROENTGEN FINDINGS

Forty-seven patients (24 males and 23 females) from a group of 61 were selected for study because of the availability of their roentgenograms. The average age of patients with roentgenographic lesions was three years less than for the group as a whole (Table II).

Originally, the diagnosis of hyperparathyroidism was made only when the presence of generalized osteitis fibrosa was determined roentgenologically, or by bone biopsy of advanced lesions. Following the report of Albright *et al.*,[3] the importance of renal complications was emphasized in the early diagnosis of the disease. Refinements in laboratory methods have further decreased the incidence of skeletal lesions detected by ordinary roentgenographic methods. The reported incidence of bone lesions has varied considerably; the general consensus of opinion ranges from about one-third to 40 per cent[4,5,15,22] In most reports, the extent of skeletal surveys was not described.

In the group reported here, 13 of 47 patients (28 per cent) had bone lesions detected roentgenologically (Table III). Considering only extensive skeletal surveys that included roentgenograms of the hands, long bones, skull, and pelvis, 13 patients of a total of 28 (46 per cent) showed bone

TABLE III

INCIDENCE OF BONE INVOLVEMENT

	No. of Cases	Bone Involvement
Total number of patients examined	47	13 (28%)
Patients with complete skeletal roentgen study	28	13 (46%)
Patients with roentgen study of hands	36	13 (36%)

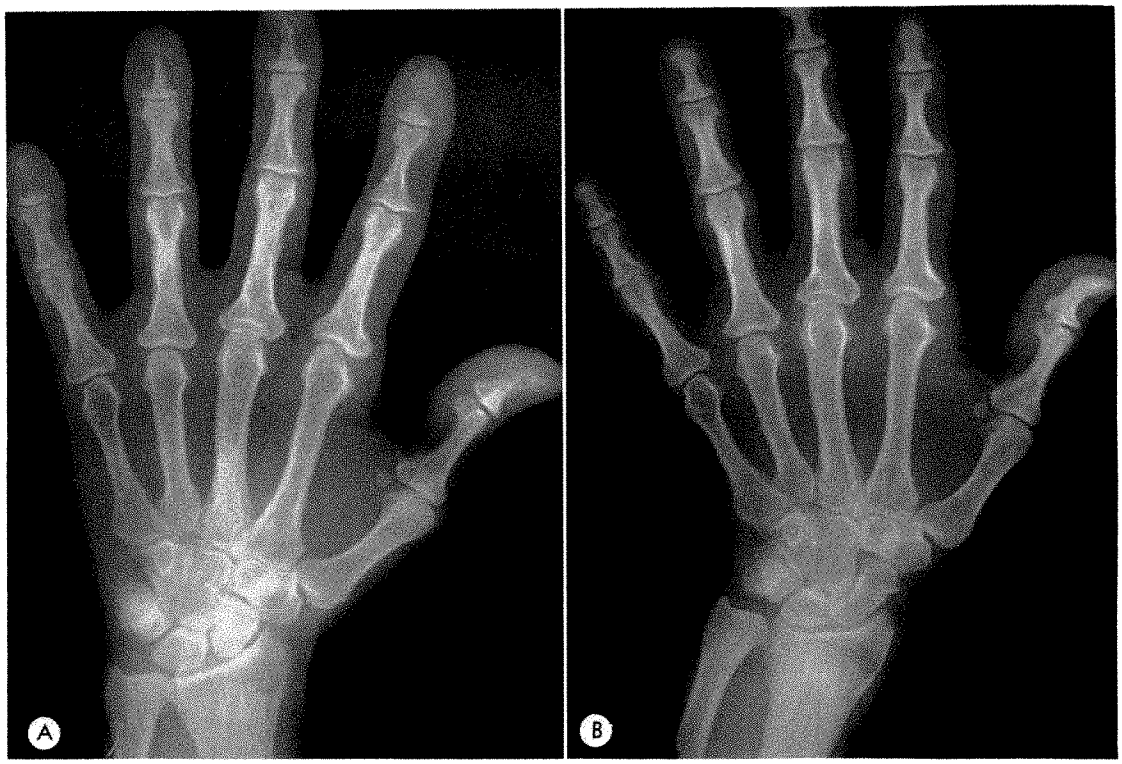


Fig. 2. (*A*) Advanced bone lesions with cortical erosion, most obvious along the lateral (thumb) aspect of the middle phalanges. The shafts of the middle phalanges have become narrow. Destructive lesions of the outer cortical surface are also visible in the proximal phalanges, metacarpals, and the distal lateral surface of the radius and medial surface of the ulna. The cortex of the sesamoid bones is rough. Extensive destruction of the shafts of the distal phalanges and of the terminal tufts has produced shortening of the terminal segments of the fingers, clinically resembling "clubbing." A cyst-like destructive lesion is present in the head of the fifth metacarpal. Irregular decalcification exists. (*B*) The same hand following parathyroidectomy. Despite healing, bone texture is not normal. Bones previously involved by destruction are now more dense than normal. The large destructive lesion in the fifth metacarpal head has not healed, indicative of a cyst.

lesions. In our experience, all patients with roentgenographic evidence of generalized osteitis fibrosa had characteristic lesions in the middle phalanges of the hands. Of the 36 patients who had roentgenograms of the hands, the incidence of 36 per cent probably represents the true incidence of bone lesions in the entire series (Table III). Of the 41 patients examined since 1954, 10 showed roentgenographic evidence of bone lesions (24 per cent).

In the classic case of hyperparathyroidism, roentgenograms show erosion of the cortical surfaces of bones, demineralization of bones, localized destructive lesions, and calcification of the soft tissues.[6,22]

Cortical erosion. In advanced cases of hyperparathyroidism, all of the cortical surfaces may be involved. In certain sites of predilection, however, cortical erosion may be visible before other manifestations of the disease. The sites most frequently involved are the lateral (radial) aspect of the middle phalanges of the hands and the tufts of the distal phalanges (Fig. 2*A*). In patients with minimal involvement, erosion is seen only near the phalangeal base in the transitional zone between the wide epiphyseal end and the slender shaft (Fig. 3). Other regions where early subperiosteal cortical erosion may be present are the articulations of the distal end of the clavicle (Fig. 4), the symphysis pubis, and the sacroiliac joints. Similar erosions may be



Fig. 3. Localized erosion involving the lateral cortical surfaces of the third and fourth middle phalangeal bases (arrows). The bones do not otherwise appear abnormal.

noted in the lamina dura (the cortical bone surrounding the root of the tooth), the calcaneal tendon insertions at the plantar or Achilles surfaces (Fig. 5), the medial aspect of the proximal end of the tibia (Fig. 6), the ischial tuberosities (Fig. 7*A*), the greater and lesser trochanters, and the crests of the ilia. Cortical erosion of the middle phalanges was present in all 13 patients with bone disease (Table IV).

The subperiosteal cortical erosion has the same roentgenologic appearance in different bones. The normal, smooth outer

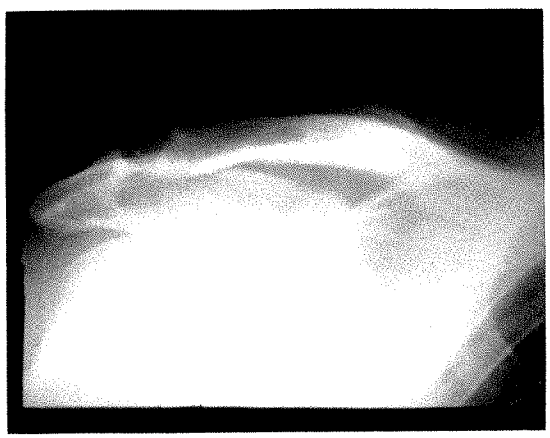


Fig. 4. Erosion of the distal end of the clavicle and adjacent surface of the acromion process.

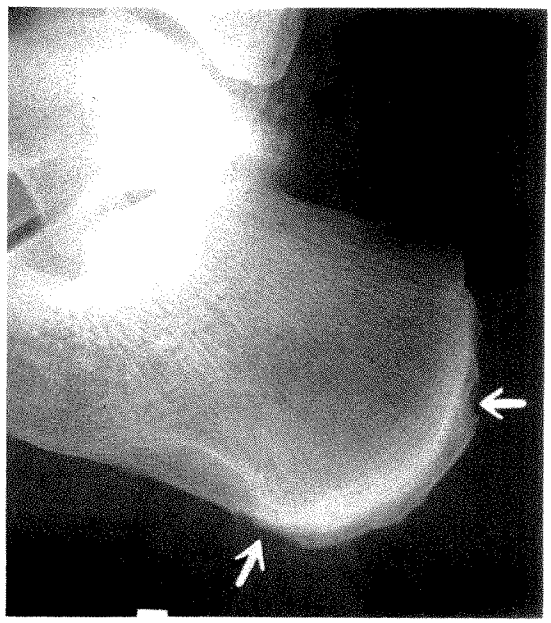


Fig. 5. Erosion of the calcaneus at the attachment of the plantar fascia and, to a lesser extent, at the Achilles tendon (arrows). These lesions, along with those of the clavicle, sacroiliac joints, symphysis pubis, and ischial tuberosities, resemble the lesions of these same structures seen in ankylosing spondylitis, rheumatoid arthritis, and Reiter's disease.

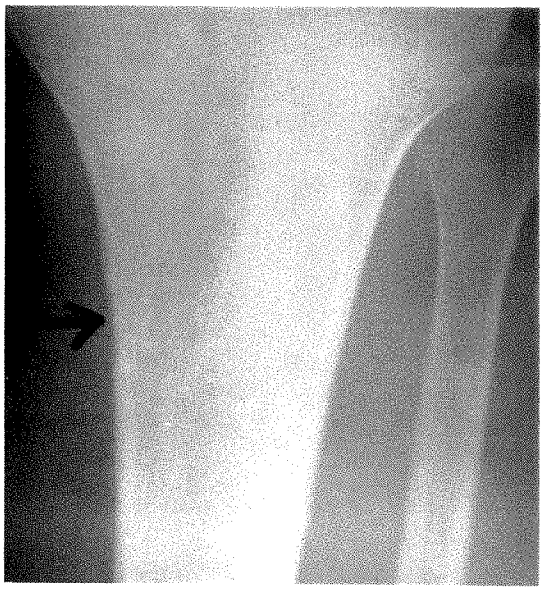


Fig. 6. A localized defect (arrow) in the upper medial cortex of the tibia is a common manifestation of hyperparathyroidism. The lesion may involve a longer segment of the tibia, but is always shallow. It corresponds in position to the attachment of the collateral tibial ligament.

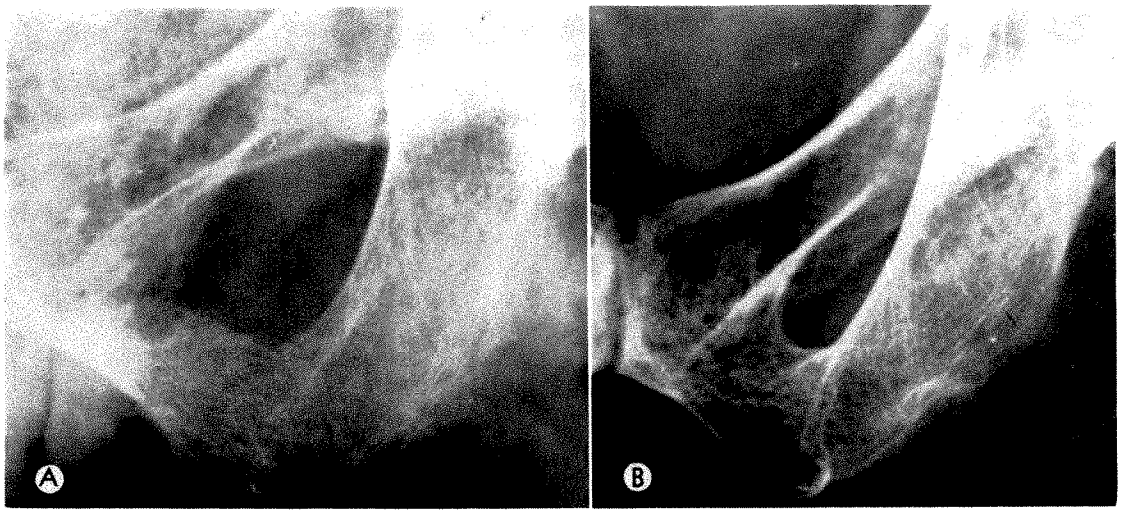


Fig. 7. (*A*) The lateral inferior cortex of the ischium and ischial tuberosity has a loss of its compact cortical surface. (*B*) Eight years after parathyroidectomy, the cortex has been restored.

surface of the cortex at first loses its sharp demarcation from the surrounding soft tissues. Later, areas of destruction alternating with normal appearing bone may produce spicules perpendicular to the cortical surface. In tubular bones, these changes are associated with vertical striations of the middle and deeper layers of the cortex caused by a widening of the haversian canals, the longitudinal spaces enclosing the blood vessels and surrounding connective tissue (Fig. 8*A*). Such typical subperiosteal resorption occurs in no other disease; when demonstrated in cases of osteomalacia due to gastrointestinal malabsorption or renal tubular dysfunction, or in cases of renal glomerular failure or pseudohypoparathyroidism, it is the result

Table IV

SITES OF BONE INVOLVEMENT

Age	Sex	Cortical Erosion of Phalanges of Hands	Absence of Lamina Dura	Cysts	Skull Mottling	Demineralization
49	F	+		○	+	+
36	F	+	+	+	+	+
41	F	+	+	+	+	+
17	F	+	+	+	○	+
64	F	+		+	○	+
36	F	+	○	○	○	○
56	M	+	○	+*	○	○
29	M	+	○	+	+	○
64	M	+	○	+†	○	+
23	M	+	○	+*	+	○
51	M	+	Upper + Lower ○	+*		+
44	M	+		+	+	+
41	M	+		+		+

* Mandible.
† Maxilla.

of secondary hyperparathyroidism.[9,17,23] Lesions resembling those of hyperparathyroidism may involve the calcaneus, sacroiliac joints, symphysis pubis, ischial tuberosity, and lateral clavicle in cases of ankylosing spondylitis, rheumatoid arthritis and Reiter's disease, but in these diseases the phalanges are not affected.

Demineralization of the skeleton. This is not always visible on roentgenograms, even in the presence of other evidence of bone involvement. Of the 13 patients in this series with bone involvement, the bones of 9 showed diminished density (Table IV). The demineralization usually differs from that of osteoporosis and osteomalacia in that it is not homogeneous. In hyperparathyroidism, bone of diminished density alternates with bone of apparently normal density, producing a mottled or striated appearance. This may be most obvious in the calvarium (Fig. 9*A*) but is also visible in other bones. Ultimately, when demineralization is extensive, it may appear homogeneous.[11] In the present series there were 10 patients with generalized homogeneous decalcification without evidence of subperiosteal resorption or destructive lesions (Fig. 10). All were middle-aged or elderly, and 8 were women. Following parathyroidectomy, roentgenograms showed that in these patients demineralization remained, and was

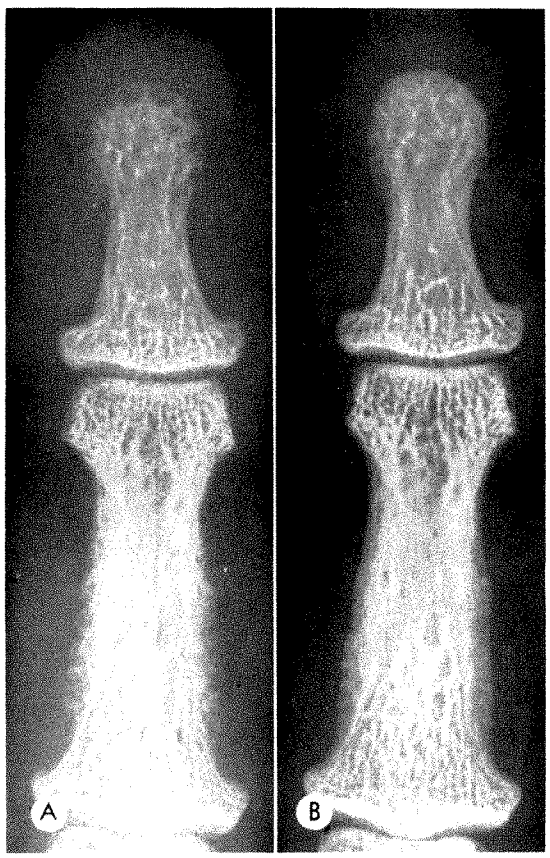


FIG. 8. (*A*) Extensive cortical destruction of the middle phalanx with horizontal spiculation. Vertical striations are prominent due to erosion of the walls of the haversian canals. Some bone resorption of the distal phalangeal tuft is visible. (*B*) Three months after parathyroidectomy, the periosteum has deposited a continuous sheet of bone, filling in the spaces between the horizontal spicules.

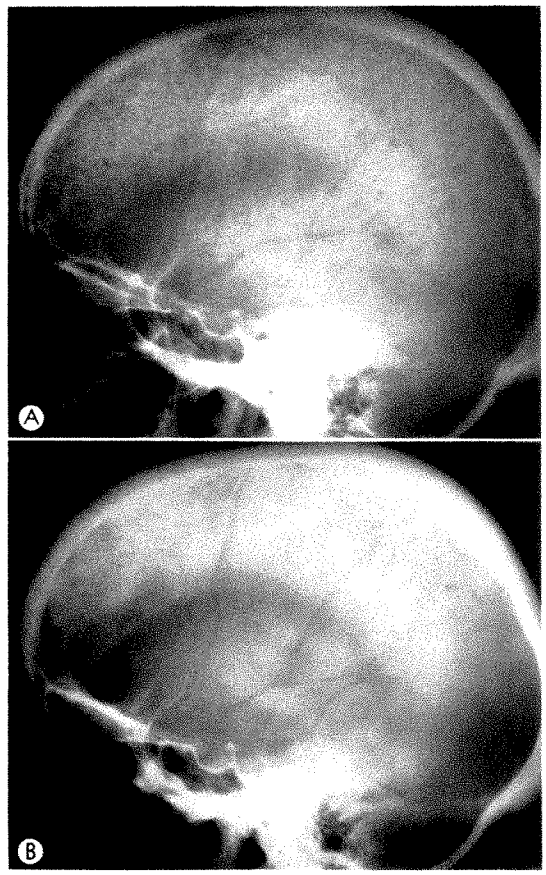


FIG. 9. (*A*) The calvarium has a mottled appearance along its periphery, most obvious distal to the inferior temporal line. The grooves for the peripheral branches of the meningeal vessels are less obvious than in *B*. (*B*) Seven years following parathyroidectomy, the calvarium is more dense.



Fig. 10. In a sixty-eight year old female with diffuse homogeneous demineralization of all bones, the cortex is thin but sharply defined. In roentgenograms after parathyroidectomy, no change in mineralization of the skeleton was apparent, suggesting that the visible bone disease was osteoporosis rather than osteitis fibrosa.

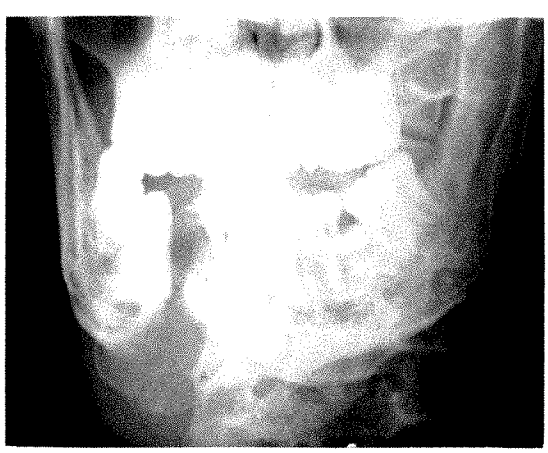


Fig. 11. "Brown tumor" focus in the right side of the mandible. The lesion has extended beyond the normal periosteal margin of the mandible and is enclosed in a thin layer of periosteal bone. The lamina dura, as seen in dental roentgenograms, was intact.

probably the result of postmenopausal or senile osteoporosis. Pugh[21] also noted diffuse demineralization without evidence of osteitis fibrosa in some patients and concluded that they may have had osteoporosis. Others have found demineralization of the skeleton, presumably from hyperparathyroidism, in the absence of other evidence of hyperparathyroid bone disease.[11,15]

Localized destructive lesions. Also known as "brown tumors," these are frequently found in the jaws or facial bones, but may be present in any part of the skeleton. In the shaft of a long bone they may be eccentric. Roentgenographically, they are usually sharply demarcated and may expand the cortex (Fig. 11). A single lesion of one bone, or many lesions involving several bones, may be present. In 11 patients with destructive lesions there was subperiosteal resorption of the middle phalanges of the hands, but the surrounding bone did not appear demineralized in 3 (Table IV). The "brown tumors" heal after removal of the parathyroid tumor, or after local radiation therapy or curettage. Some undergo cystic degeneration, persisting as defects in the bone after the primary disease has been cured. The cystic lesions do not differ roentgenographically from the tumors and can be recognized only by their failure to heal (Fig. 2*B*). All 4 patients in this series with localized destructive lesions of the jaw had persistence of the lamina dura (Table IV). In some cases the tumor encroached upon the adjacent lamina dura, producing a localized destruction but with the lamina dura of the other teeth remaining intact (Fig. 12). One patient had a tumor of the mandible with absence of the lamina dura but with normal lamina dura in the maxilla.

The degree of bone involvement compared with blood chemical determinations. This is shown in Table V. Serum calcium and phosphorus determinations were obtained in all 13 patients. No correlation could be found between the values of these chemical determinations and the presence or absence of bone involvement. The serum alkaline



Fig. 12. Destructive lesion ("brown tumor") involving the cancellous bone about the roots of the right lower cuspid and first bicuspid. The lamina dura is destroyed in the region of the tumor but is intact elsewhere.

phosphatase, however, was a reliable guide to the presence or absence of osteitis fibrosa and an index to the extent of the bone disease. The alkaline phosphatase in patients without roentgenologic evidence of bone lesions ranged from 3.0 to 7.7 Bodansky units and averaged 5.4. Two patients with values of 9.0 and 25.5 Bodansky units were eliminated because of the presence of Paget's disease, which also produces an elevation in the alkaline phosphatase. In patients with roentgenologic evidence of

Table V

DEGREE OF BONE INVOLVEMENT IN RELATION TO BLOOD CHEMICAL DETERMINATIONS

Age	Sex	Bone Involvement	Alkaline Phosphatase (Bodansky Units)	Calcium (mg. per 100 cc.)	Phosphorus (mg. per 100 cc.)
64	M	Minimal	5.0	14.4	2.4
36	F		6.2	14.2	3.0
41	M		11.0	16.4	2.3
49	F	Moderate	8.0	15.4	3.5
51	M		11.0	14.5	1.1
64	F	Severe	11.0	12.2	1.8
23	M		17.7	16.0	1.7
17	F		33.2	17.6	2.6
51	M		35.0	12.0	1.4
44	M		36.5	14.5	2.7
41	F		46.0	13.9	2.4
29	M		56.0	15.4	3.0
36	F			15.6	2.2

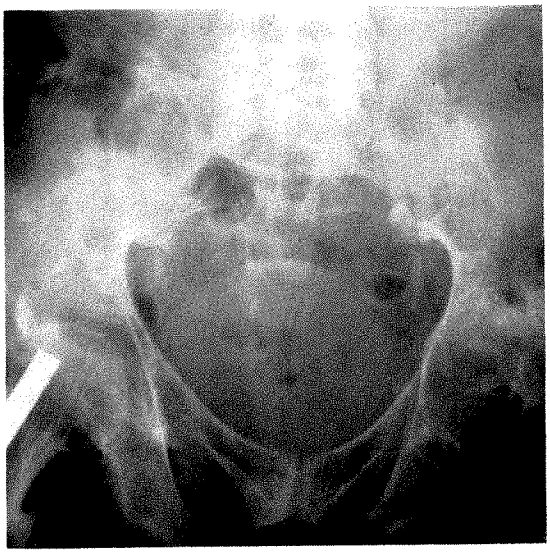


FIG. 13. A roentgenogram several years after parathyroidectomy demonstrates healed bone lesions. Bone bridges the symphysis pubis and sacroiliac joints. Bone extends from the ischial tuberosities and lesser trochanter of the right femur into the adjacent soft tissues. On preoperative roentgenograms, destruction of cortical bone was visible at these sites.

bone disease, the range was from 5.0 to 56.0 Bodansky units and the average was 23.1. These patients were divided into three groups, depending on the extent of bone disease. The serum alkaline phosphatase varied directly with the severity of the bone disease. Only 1 patient had an alkaline phosphatase determination approaching the normal value (normal 2.0 to 4.5 Bodansky units). A similar correlation has been noted by others.[21]

Healing of bone lesions. In one patient, roentgenographic studies repeated for four years showed slight progression of the bone lesions. Although we have not seen any cases of spontaneous regression, clinically intermittent hyperparathyroidism has been reported.[25] Rapid healing follows parathyroidectomy, with improvement noted on roentgenograms within four to six weeks. The recalcification begins at the periphery of the lesion. In areas of cortical resorption, calcified bone first becomes visible as a thin dense linear shadow parallel to the cortex but separated from it by the intervening fibrous and osteoid tissue. Subsequently, this radiolucent tissue becomes converted to dense bone (Fig. 2*B*, 7*B*, 8*B*, and 9*B*). The localized destructive lesions also begin repair at the periphery with ultimate replacement by dense bone. The mottled decalcified areas become denser than in normal bone, frequently producing exaggerated striations. The newly formed bone may extend along tendons or ligaments, which may result in fusion of the symphysis pubis and sacroiliac joints (Fig. 13). In our experience, the bones usually do not revert to normal but continue to harbor the dense scars or radiolucent cysts. That this bone is susceptible to trauma is demonstrated by 1 of our patients whose jaw was fractured during a dental extraction ten years after parathyroidectomy.

Localized roentgen therapy to the bone lesion also induces healing with the same pattern as that which follows parathyroidectomy.

Kidney lesions. Roentgenologic evidence of urinary calculi was present at one time or another during the course of the disease in 36 (77 per cent) of the patients (Table VI). Defining nephrocalcinosis as diffuse calcification of the renal parenchyma, 4 patients fitted into this category, whereas 32 had discrete renal calculi. The renal calculi varied in size and density, were unilateral or bilateral, and single or multiple. Hellström,[11] defining nephrocalcinosis as multiple small calcified areas in the renal parenchyma, either localized or diffuse, found nephrocalcinosis in 37 of a total of 56 patients. It is known from renal biopsy or autopsy that nephrocalcinosis is present in many patients in whom it could not be detected roentgenographically. Many patients reported having or passing calculi for several years before the diagnosis of hyperparathyroidism was established. All but 1 of the 13 patients with bone disease had renal calculi during the course of the illness. Four of the patients had calculi prior to the onset of bone lesions but not roentgenographically evident at the time bone lesions appeared.

Soft tissue calcification. Except for calcification of the kidneys or conjunctiva, this is not a common feature of the disease. It is present only in severe cases with extensive renal damage. Considerable vascular calcification was noted in 2 patients who had bone lesions and uremia. One patient, without bone or renal disease, had calcifications of the articular cartilages of one wrist which were probably not related to the disease. Another patient, sixty-seven years old, with renal calculi but no bone lesions, had extensive vascular calcification, perhaps the result of arteriosclerosis.

The weight of the parathyroid tumor related to the presence of bone or kidney lesions. Involved parathyroid glands in patients with bone disease averaged more than five times the weight of those in cases without bone involvement (Table VII).

A similar relationship was noted by Woolner *et al.*[26] and Hellström.[13] Hellström reported that the size of the parathyroid neoplasm was not related to the duration of symptoms. In patients with renal disease, he found that small adenomas with a large number of "wasserhelle" cells predominated; in those with skeletal lesions, large adenomas with few "wasserhelle" cells were the rule.

Esophageal and tracheal displacement. Thirty patients had roentgenograms of the pharynx and upper esophagus, 3 showing displacement of the esophagus and 3 of the trachea by an adjacent soft tissue mass. Four of these 6 patients, however, also had thyroid nodules, one definitely causing the tracheal displacement. In the entire series,

TABLE VI

INCIDENCE OF RENAL CALCIFICATION

	No. of Cases	Male	Female
Total number of patients with renal calcification	36 (77%)	20	16
Patients with nephrocalcinosis	4	2	2
Patients with renal calculi	32	18	14

TABLE VII

WEIGHT OF INVOLVED PARATHYROID GLANDS

	Range (gm.)	Average (gm.)
With bone disease	1.0 –11.8	5.02
Without bone disease	0.074– 5.0	0.94

13 patients (28 per cent) had thyroid neoplasms. Four of these were carcinomas and 9 were thyroid adenomas or involutional goiters. The high incidence of coexisting neoplastic changes in the thyroid and parathyroid glands has been reported by others.[13] Wyman and Robbins[27] detected parathyroid adenomas in 20 or 30 patients who were given adequate examinations. However, 23 patients were excluded from their series because the examination had been inadequate or because roentgenograms were not available. They also disregarded the possibility that associated thyroid neoplasms caused the esophageal or tracheal displacement. The lesions most likely to produce displacement of adjacent structures are those of large size, usually easily identified by the surgeon. Despite inherent pitfalls in the interpretation of roentgenograms of the neck and upper mediastinum for location of parathyroid masses, roentgenograms are sometimes of considerable value.

DISCUSSION

The most important roentgenograms for the diagnosis of generalized osteitis fibrosa are those of the hands in the posteroanterior view. In the present series, all patients with roentgenographically demonstrable bone lesions had subperiosteal erosion of the lateral (radial) aspect of some of the phalanges and almost all had erosion of the cortex of the distal phalangeal tufts of one or more fingers. In some cases, not all of the middle phalanges were involved, and, occasionally, the lesions were more obvious in one hand than in the other. Subperiosteal resorption is pathognomonic of generalized osteitis fibrosa, whether it is the

result of primary or secondary hyperparathyroidism. Cases of osteomalacia with mottling of the skull, absence of the lamina dura, subperiosteal resorption, and cyst-like destructive lesions have been described, but these cortical changes are the result of secondary hyperparathyroidism.[9,23]

A review of the literature reveals an overwhelming emphasis on the role of the disappearance of the lamina dura (the cortical bone lining the tooth socket) in the diagnosis of hyperparathyroid disease. Two other lesions of the jaw bones, demineralization and pseudocysts (giant cell osteoclastomas), have been described. It has frequently been reported that one or more of these changes occur in the mandible and maxilla in about half the patients with this disease. Unfortunately, the reported series were small and not well substantiated, with the evidence apparently derived from selected patients. Furthermore, many of the dental changes were based on lateral jaw and skull roentgenograms, which are of little or no value in interpreting the fine trabecular and lamina dura changes.

Of the 13 patients with roentgenologic evidence of bone lesions, 9 dentulous patients had adequate dental roentgenographic examinations. Only 4 of these had absence of the lamina dura. In a recent series of 29 consecutive dentulous patients with histologically proved parathyroid adenomas (including some from this investigation and others which were seen more recently) 2 had, in addition to other skeletal lesions, destruction of the mandible and maxilla. One of these patients had partial loss of the lamina dura about two teeth (Fig. 12). None of the other 27 patients showed abnormal bone changes in dental roentgenograms, although 10 had roentgenographic evidence of disease in other bones.

These observations suggest that, when applied as a sensitive reflection of parathyroid status, jaw bone changes in dental roentgenograms have been overemphasized.

Erosion of the endosteal, cortical surface of bones probably is as advanced, or more so, than that on the periosteal surface and may be observed on some roentgenograms.[16] Its detection is more difficult because of the overlying bone projected upon the roentgenograms and because the normal endosteal bone is less compact and the contour is irregular.

Secondary hyperparathyroidism is an interesting complication following surgical removal of a functioning parathyroid adenoma. The roentgenologic manifestations have not been previously reported, probably because routine postoperative roentgenograms of the hands at frequent intervals are not ordinarily obtained. In 1 of our patients, it was interesting to compare a preoperative roentgenogram (Fig. 14*A*) with a postoperative roentgenogram (Fig. 14*B*) made four months following the surgical procedure. The considerable healing which was seen coincided with a depressed serum calcium (from 12.5 mg. per 100 cc. to 8.5 mg. per 100 cc.) and no appreciable change in the urinary phosphorus (3.1 to 3.4 mg. per 100 cc.). Two and one-half months later (Fig. 14*C*), the destructive bone lesions were more advanced, associated with a drop in the serum calcium to 7.8 mg. per 100 cc. Subsequently, the serum calcium rose to normal values and the bone lesions demonstrated a continuing improvement. The patient had been maintained on a low calcium diet during the pre- and postoperative course because of nephrocalcinosis and moderately impaired renal function. The course of the renal disease did not change following parathyroidectomy.

A problem of concern is that of the frequent disparity in involvement of kidneys and bones in some patients. Albright and Reifenstein[2] expressed the opinion that high calcium diets favor renal disease and prevent bone disease. To substantiate this theory, they demonstrated healing of bone lesions by increased calcium in the diet. Dent[10] observed the same improvement after vitamin D treatment, which is in agreement with our experience.

In the present investigation, the initial

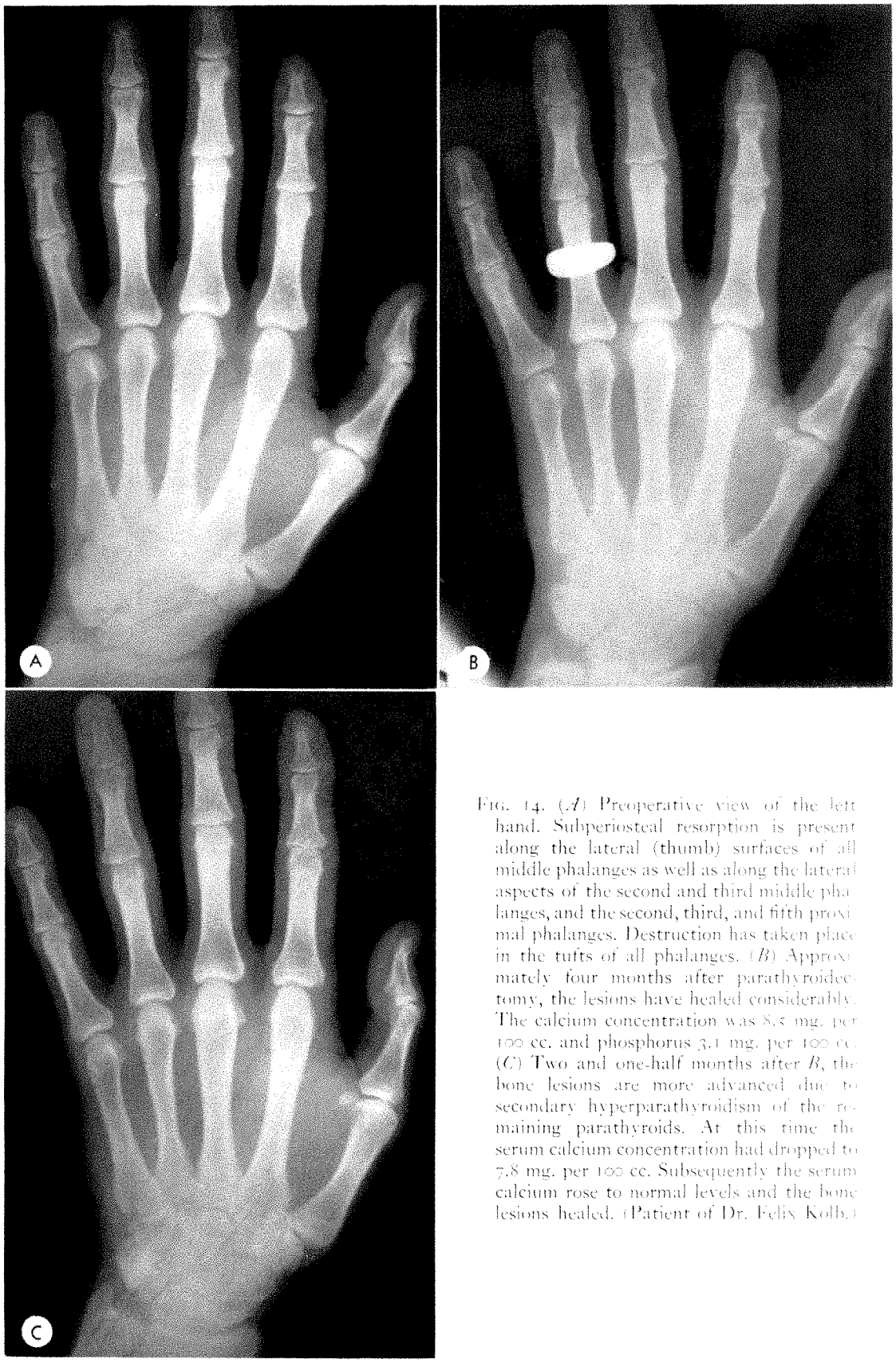


FIG. 14. (*A*) Preoperative view of the left hand. Subperiosteal resorption is present along the lateral (thumb) surfaces of all middle phalanges as well as along the lateral aspects of the second and third middle phalanges, and the second, third, and fifth proximal phalanges. Destruction has taken place in the tufts of all phalanges. (*B*) Approximately four months after parathyroidectomy, the lesions have healed considerably. The calcium concentration was 8.5 mg. per 100 cc. and phosphorus 3.1 mg. per 100 cc. (*C*) Two and one-half months after *B*, the bone lesions are more advanced due to secondary hyperparathyroidism of the remaining parathyroids. At this time the serum calcium concentration had dropped to 7.8 mg. per 100 cc. Subsequently the serum calcium rose to normal levels and the bone lesions healed. (Patient of Dr. Felix Kolb.)

manifestations of hyperparathyroidism in 4 patients were renal calculi without bone disease. Subsequently, the stones were passed and bone lesions developed. The stones did not re-form, even with the continued severe hyperparathyroidism. Retrospective analysis of the diets is apt to be misleading. Following the kidney lesions the patients may have adopted low calcium diets, shifting the burden from the kidneys to the bones.

However, the lack of bone lesions, despite a low mineral diet upon which most of our patients were maintained during their management and metabolic investigations, indicates that diet is not of great importance. Although some patients with severe renal disease and primary or secondary hyperparathyroidism show elevated serum phosphorus values and denser-than-normal bones on roentgenograms, characteristic subperiosteal resorption is still exhibited.

Munson's[20] evidence indicates that the reaction to parathyroid extracts by bone and kidneys can be partially differentiated and is in reality two separate actions. In patients with only a slight tendency to bone lesions, a high calcium diet may prevent their progression, and, if this is true, calcium deprivation should promote bone lesions. Further studies will determine whether bone lesions can be induced in hyperparathyroid patients maintained on a low calcium diet.

SUMMARY

Of a total of 61 patients in whom the surgically removed parathyroid gland proved to be neoplastic on histologic examination, roentgenograms of 47 were adequate for evaluation. Thirteen had roentgenographic evidence of osteitis fibrosa. All of these patients had subperiosteal cortical resorption of some of the middle phalanges of the hands.

Cortical resorption at other sites, such as at the lamina dura and tendinous insertions, was less frequently seen.

Diffuse demineralization of the skeleton was not always visible on roentgenograms even when other roentgenographic evidence of bone involvement existed. The demineralization, when present, was usually of coarse granularity or striated.

Localized destructive lesions ("brown tumors") may be present, even in the jaws, without significant resorption of the lamina dura. All patients with "brown tumors" had subperiosteal cortical resorption of the middle phalanges of the fingers.

The alkaline phosphatase level was significantly elevated in 11 of the 13 patients whose roentgenograms showed evidence of osteitis fibrosa. The extent of osseous disease correlated well with the level of serum alkaline phosphatase, but not with the serum calcium or phosphorus values.

Roentgenographic evidence of urinary calculi was present at one time or another during the course of the disease in 36 patients. Four of these patients had diffuse nephrocalcinosis.

Esophageal or tracheal displacement was observed in 6 of 30 patients who had roentgenograms of the area. This finding was of doubtful value, however, since many patients with hyperparathyroidism also have thyroid neoplasms which may cause displacement.

Howard L. Steinbach, M.D.
Department of Radiology
University of California Medical Center
San Francisco 22, California

ADDENDUM

Since this manuscript was accepted for publication, 34 additional patients with hyperparathyroidism have been studied, making a total of 95. Of the last 34 patients, 1 had two water-clear-cell adenomas of the parathyroid, 1 had endocrine adenomatosis; the other 32 each had single parathyroid adenomas. In the total sreies of 95 patients, 22 had roentgen evidence of osteitis. In 14 patients, far advanced bone disease with multiple cysts was present; in 7 patients, subperiosteal resorption of bone was the only evidence of hyperparathyroid bone disease; in 1 patient there was no subperiosteal resorption of the pha-

langes, but patchy resorption of the lamina dura was associated with a histologically proved giant cell tumor of the mandible. This was the only 1 of 16 dentulous patients examined in the additional 34 patients who showed any dental abnormalities attributable to hyperparathyroidism. The total incidence of osteitis fibrosa demonstrated on roentgenograms in the entire 95 patients was 23 per cent.

REFERENCES

1. ALBRIGHT, F., AUB, J. C., and BAUER, W. Hyperparathyroidism; common and polymorphic condition as illustrated by seventeen proved cases from one clinic. *J.A.M.A.*, 1934, *102*, 1276–1287.
2. ALBRIGHT, F., and REIFENSTEIN, E. C., JR. The Parathyroid Glands and Metabolic Bone Disease. Williams & Wilkins Co., Baltimore, 1948.
3. ALBRIGHT, F., BAIRD, P. C., COPE, O., and BLOOMBERG, E. Studies on physiology of parathyroid glands; renal complications of hyperparathyroidism. *Am. J. M. Sc.*, 1934, *187*, 49–65.
4. BLACK, B. M. Hyperparathyroidism. Charles C Thomas, Springfield, 1953.
5. BOGDONOFF, M. D., WOODS, A. H., WHITE, J. E., and ENGEL, F. L. Hyperparathyroidism. *Am. J. Med.*, 1956, *21*, 583–595.
6. CAMP, J. D. Osseous changes in hyperparathyroidism; roentgenologic study. *J.A.M.A.*, 1932, *99*, 1913–1917.
7. CHAMBERS, E. L., JR., GORDAN, G. S., GOLDMAN, L., and REIFENSTEIN, E. C., JR. Tests for hyperparathyroidism: tubular reabsorption of phosphate, phosphate deprivation, and calcium infusion. *J. Clin. Endocrinol.*, 1956, *16*, 1507–1521.
8. COPE, O., KEYNES, W. M., ROTH, S. I., and CASTLEMAN, B. Primary chief-cell hyperplasia of parathyroid glands: new entity in surgery of hyperparathyroidism. *Ann. Surg.*, 1958, *148*, 375–388.
9. DAVIES, D. R., DENT, C. E., and WILLCOX, A. Hyperparathyroidism and steatorrhoea. *Brit. M. J.*, 1956, *2*, 1133–1137.
10. DENT, C. E. Personal communication.
11. ELLIS, K., and HOCHSTIM, R. J. Skull in hyperparathyroid bone disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 732–742.
12. HARRISON, H. E., and HARRISON, H. C. Renal excretion of inorganic phosphate in relation to action of vitamin D and parathyroid hormone. *J. Clin. Invest.*, 1941, *20*, 47–55.
13. HELLSTRÖM, J. Primary hyperparathyroidism; observations in a series of 50 cases. *Acta endocrinol.*, 1954, *16*, 30–58.
14. HELLSTRÖM, J. Calcification and calculus formation in a series of 70 cases of hyperparathyroidism. *Brit. J. Urol.*, 1955, *27*, 387–393.
15. HELLSTRÖM, J. Experience from one hundred and five cases of hyperparathyroidism. *Acta chir. scandinav.*, 1957, *113*, 501–505.
16. JAFFE, H. L., BODANSKY, A., and BLAIR, J. E. Influence of age and of duration of treatment on production and repair of bone lesions in experimental hyperparathyroidism. *J. Exper. Med.*, 1932, *55*, 139–154.
17. KOLB, F., and STEINBACH, H. L. Syndrome of pseudo-hypo-hyperparathyroidism. *Acta endocr.* (Kbh), 1960, Suppl. 51, p. 475.
18. MCLEAN, F. C., and URIST, M. R. Bone: An Introduction to the Physiology of Skeletal Tissue. University of Chicago Press, Chicago, 1955.
19. MCLEAN, F. C. Ultrastructure and function of bone. *Science*, 1958, *127*, 451–456.
20. MUNSON, P. L. Studies on role of parathyroids in calcium and phosphorus metabolism. *Ann. New York Acad. Sc.*, 1955, *60*, 776–796.
21. PUGH, D. G. Subperiosteal resorption of bone; roentgenologic manifestation of primary hyperparathyroidism and renal osteodystrophy. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *66*, 577–586.
22. PUGH, D. G. Roentgenologic diagnosis of hyperparathyroidism. *S. Clin. North America*, 1952, *32*, 1017–1030.
23. STEINBACH, H. L., KOLB, F. O., and CRANE, J. T. Unusual roentgen manifestations of osteomalacia. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1959, *82*, 875–886.
24. WEINMANN, J. P., and SICHER, H. Bone and Bones. Second edition. C. V. Mosby Co., St. Louis, 1955.
25. WHITBY, L. G. Intermittent hyperparathyroidism. *Lancet*, 1958, *1*, 883–885.
26. WOOLNER, L. B., KEATING, F. R., JR., and BLACK, B. M. Tumors and hyperplasia of parathyroid glands; review of pathological findings in 140 cases of primary hyperparathyroidism. *Cancer*, 1952, *5*, 1069–1088.
27. WYMAN, S. M., and ROBBINS, L. L. Roentgen recognition of parathyroid adenoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1954, *71*, 777–784.

ROENTGEN CHANGES IN REITER'S SYNDROME

By WILLIAM V. WELDON, CAPT., MC, USA,* *and*
RAYMOND SCALETTAR, CAPT., MC, USA†
WASHINGTON, D. C.

REITER's syndrome is a triad of urethritis, arthritis, and conjunctivitis. In 1818 and 1836 Brodie[1] reported 6 case histories of men who had this triad of symptoms. In 1916 Reiter[9] described the symptom-complex in patients with dysentery. Accounts of the postdysenteric form of the syndrome, as reported in Europe, indicate that it has the same characteristics and clinical course as the venereal form. The syndrome occurs almost exclusively in the male and is of unknown etiology.

Although the triad of symptoms has been emphasized, many patients also have diarrhea as an early symptom, and others have mucocutaneous lesions. Patients with nonspecific urethritis and arthritis alone have a clinical course indistinguishable from the complete syndrome. Many investigators[2,10] believe that nonspecific urethritis with arthritis is an incomplete form of the fully developed syndrome; therefore, these cases are included in this study. The following features are reported because of their special roentgenologic interest.

Sixty-four patients with Reiter's syndrome, or nonspecific urethritis associated with arthritis, were treated at Walter Reed General Hospital from 1950 to 1960. The available roentgenograms of 46 patients were reviewed to determine what roentgen changes might suggest the syndrome. The evidence collected indicates that the syndrome has a consistent roentgenologic pattern.

ROENTGEN FINDINGS

Acute polyarthritis dominated the clinical picture, although occasionally a single joint was involved. The predilection for the joints of the lower extremities and sacro-iliac joints is found in all large series (Table I).

The frequency and distribution of roentgenographic changes are shown in Table II. Characteristic changes were most frequently found in the bones and joints of the feet, ankles, and in the sacro-iliac joints. The knees were often involved but with no characteristic findings. The hips and shoulders were less commonly involved. In only a few cases were changes in the elbow, wrist, phalanges of the hand, symphysis pubis, and sternoclavicular joint found. Clinical involvement of the feet, ankles, knees, and sacro-iliac joints was associated with roentgenographic changes to a greater degree than in other joints. In many cases the only changes noted were peri-articular swelling and subchondral bone resorption. Other cases progressed within a period of weeks or months to the characteristic changes of periostitis and bony erosion.

Most cases in which severe roentgenographic changes occurred were in patients with an illness lasting several months or those with repeated attacks over a period of many months.

Roentgenograms taken early in the course of the illness seldom showed more than peri-articular swelling and slight subchondral resorption of the adjacent bones. Occasionally, joint effusion was noted, especially in the knee. Some patients respond to treatment and the disease never progresses beyond this phase. When symptoms progressed, with limitation of motion of the involved joints, patchy and frequently marked diffuse demineralization was observed. These findings were most common in the metatarsophalangeal joints, ankles, knees, and interphalangeal joints of the feet and hands. These roentgenographic

* Radiology Service, Walter Reed General Hospital, WRAMC, Washington, D. C.
† Department of Medicine, Walter Reed General Hospital, WRAMC, Washington, D. C.

TABLE I

FREQUENCY AND DISTRIBUTION OF JOINTS CLINICALLY INVOLVED

Upper Extremity		Head and Trunk		Lower Extremity	
	Per Cent		Per Cent		Per Cent
Fingers	14	Sternoclavicular	5	Hip	28
Wrist	16	Lumbar and sacro-iliac	42	Knee	67
Elbow	12	Temporomandibular	2	Ankle	65
Shoulder	21			Tarsal and metatarsals	65
				Toes	44

changes were usually reversible. Although some patients showed only the usual reversible changes of peri-articular swelling and demineralization, many had severe clinical joint symptoms with repeated attacks resulting in bony cortical erosion, periosteal new bone formation, and joint destruction. In this chronic stage of the illness, the characteristic fluffy prolific periosteal new bone formation was noted on the plantar surface of the calcaneus. Marginal erosions and loss of the joint space with marked demineralization were noted in some cases, especially in the metatarsophalangeal and interphalangeal joints of the feet. There were no cases of ankylosis, and the only significant deformity observed was the painful calcaneal spur.

Erosion of the articular surfaces was most often seen in the small joints of the feet, knees, and sacro-iliac joint. The earliest erosions were small circumscribed areas of the periphery of the articular surface surrounded by normal appearing bone (Fig. 1*A*). Only rarely did the erosion progress to destroy the subchondral cortex

TABLE II

FREQUENCY AND DISTRIBUTION OF ROENTGENOGRAPHIC CHANGES IN 46 PATIENTS WITH REITER'S SYNDROME

Site	No. of Patients Roentgenographed	Changes
Os calcis	20	Periostitis-10; erosion-3
Ankles	30	Soft tissue swelling-15; periostitis-1; demineralization-13; erosion-4
Tarsals	38	Soft tissue swelling-3; erosion-3; demineralization-13
Metatarsals	38	Soft tissue swelling-10; periostitis-3; demineralization-15; erosion-3
Phalanges of foot	38	Soft tissue swelling-1; periostitis-5; demineralization-14; erosion-4
Hips	10	Demineralization-4; joint destruction-1
Sacro-iliac joints	32	Bilateral changes-11; unilateral changes-4; joint narrowing-9; erosion-15; sclerosis-6
Spine	6	Lateral calcific bridging-1; demineralization-11
Knee	36	Soft tissue swelling-18; periostitis-1; demineralization-11; effusion-8; erosion-4
Shoulder	10	Demineralization-2; erosion-1
Elbow	8	Soft tissue swelling-3; periostitis-2
Carpals	13	Soft tissue swelling-3; erosion-1
Metacarpals	13	No changes
Phalanges of hand	13	Soft tissue swelling-3; periostitis-1; demineralization-2
Symphysis pubis	10	Erosion and sclerosis-2
Sternoclavicular joint	2	Demineralization and erosion-2

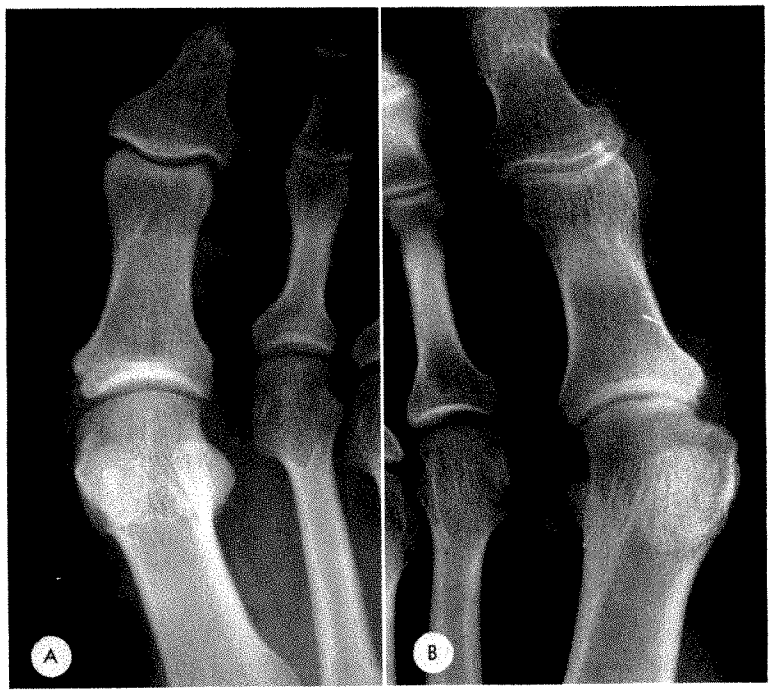


FIG. 1. (*A*) Peri-articular swelling about metatarsophalangeal joint with early juxta-articular erosion of base of proximal phalanx of great toe. The joint space is not involved at this time. (*B*) Erosion of metatarsophalangeal joint of left great toe.

of the articular surface except in the sacroiliac and metatarsophalangeal joints (Fig. 1*B*). Only one patient had severe erosion of the hip joint (Fig. 2).

Similar erosive changes occurred on the posterior and inferior aspects of the calcaneus at the attachment of the Achilles tendon and plantar fascia. These changes were always accompanied by pain or tenderness of the Achilles tendon or plantar aspect of the calcaneus. Erosive changes also occurred in the adjacent articular surfaces of the tarsals, malleoli, carpals, shoulders and knees (Fig. 3).

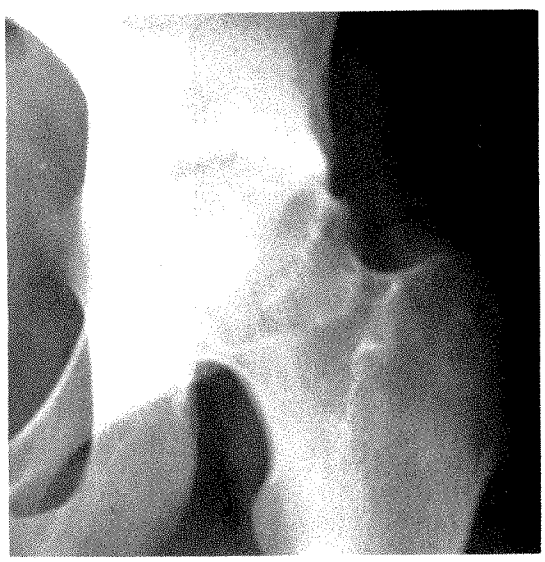


FIG. 2. Unilateral destruction of left hip joint of 22 months' duration.

Periosteal new bone formation was the most characteristic roentgenographic finding. These changes varied with the bones affected. Achilles tendinitis and plantar fasciitis frequently result in loss of associated bony cortex with subsequent bony proliferation into the adjacent soft tissue to produce calcaneal spurs. These spurs were of two types: a simple form in which the margins were well outlined and not associated with changes beyond the base of the spur; and a markedly proliferative form which was irregular in contour. The simple spur is believed to be an ossification in the attachment of the fibers of the long plantar ligament. The characteristic type



Fig. 3. Peri-articular swelling, joint effusion, severe erosion, and periosteal reaction of anterior portion of tibia. Note severe tibiofibular joint destruction.

is a more diffuse process, producing a fluffy outline on the plantar surface of the calcaneal tuberosity and beyond (Fig. 4). Mason *et al.*,[6] in a comparative study of Reiter's syndrome, rheumatoid arthritis, and ankylosing spondylitis, found simple spurs in 40 per cent of the total cases of both Reiter's syndrome and rheumatoid arthritis as compared with 24 per cent in ankylosing spondylitis. On the plantar surface, however, the incidence was significantly different. Periostitis occurred in 20 per cent of the patients with Reiter's syndrome, in 4 per cent of those with rheumatoid arthritis, and in 5 per cent of those with ankylosing spondylitis. They further noted that spurs arising from the posterior aspect were more common in Reiter's syndrome (Fig. 5). In this series the florid type periostitis, often bilateral, was seen in 8 out of 20 patients of whom roentgenograms of the calcaneus were made. The impression that this type of periostitis is probably

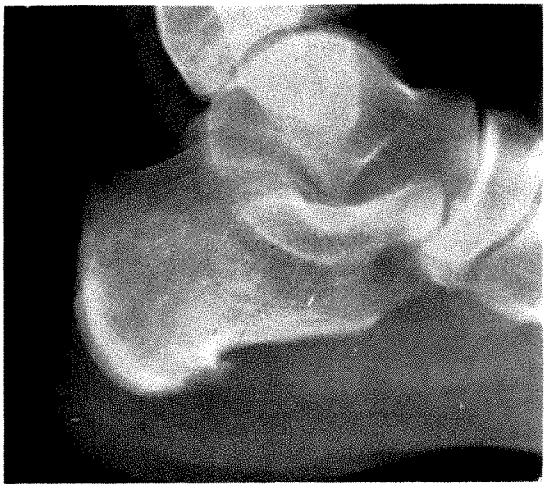


Fig. 4. Characteristic prolific spur on plantar aspect of calcaneus which developed over a period of 7 months.

pathognomonic in Reiter's syndrome is further substantiated.

The second type of characteristic periosteal new bone formation found in this series of Reiter's syndrome was that of linear streaks parallel to the cortex of both the diaphysis and metaphysis of the metatarsals and especially in the toes (Fig. 6). This form of periostitis was also noted in the proximal radius in 2 cases. Occasionally, the periostitis was lace-like in the toes and



Fig. 5. Spur formation on superior aspect of calcaneus at attachment of Achilles tendon.

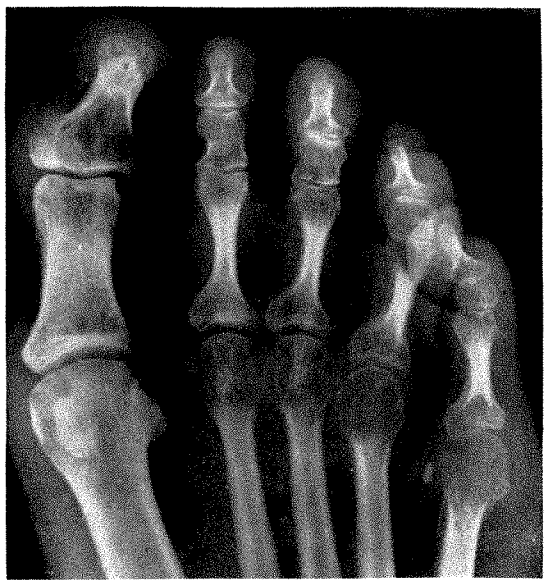


FIG. 6. Severe demineralization, peri-articular swelling, and linear periosteal reaction about metaphysis and diaphysis of third, fourth and fifth phalanges and metatarsals.

in the shaft of the fingers, much like the type seen in hypertrophic pulmonary osteoarthropathy (Fig. 7, *A* and *B*).

Although early roentgenographic changes in most joints were generally reversible, sacro-iliac joint changes were progressive. A high incidence of sacro-iliitis in Reiter's syndrome has been reported by several authors.[4,5,7,8] Fifteen of 31 patients showed roentgenographic changes in the sacro-iliac joints and these usually occurred in patients with recurrent relapses to which patients with Reiter's syndrome are so prone. The most common finding was narrowing of the joint space with irregular indistinct articular borders and frequent sclerosis (Fig. 8). Seventy-three per cent of the patients showed bilateral roentgenographic changes. Many of the findings were asymmetric.

Neither the clinical nor roentgenologic manifestations of sacro-iliac arthritis are

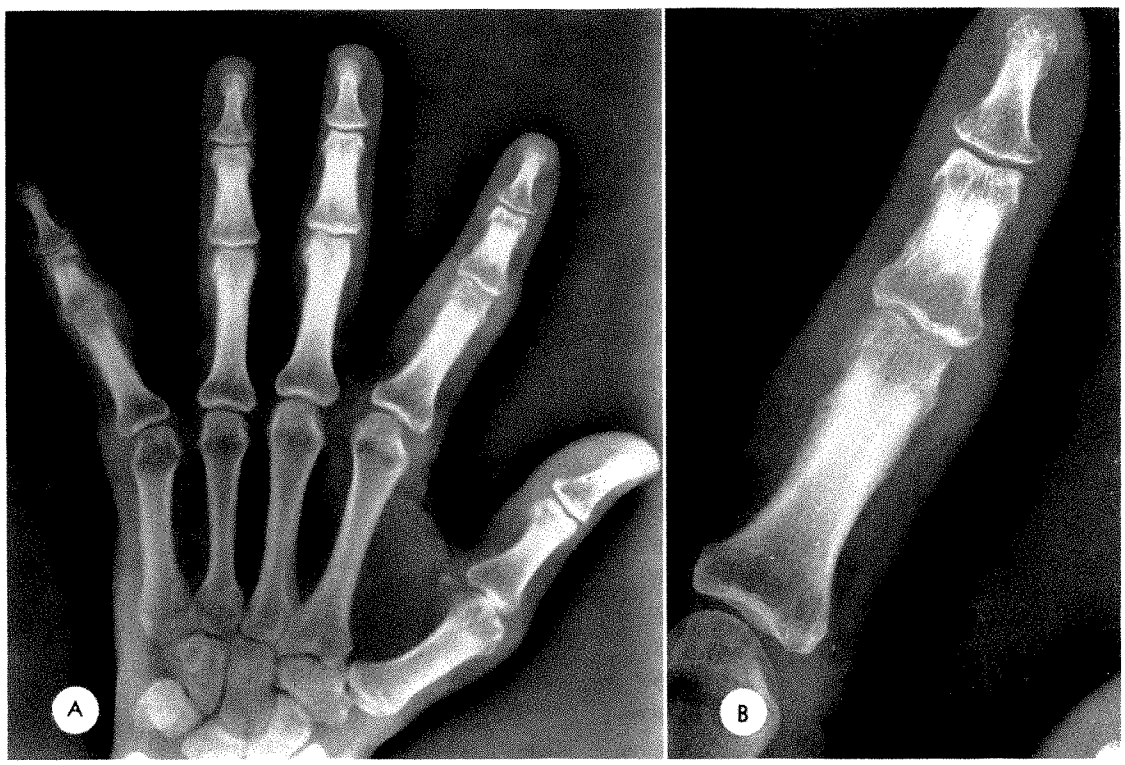


FIG. 7. (*A*) Marked peri-articular swelling about proximal interphalangeal joints of first, second and fifth digits with periosteal reaction about diaphyses. Note absence of joint involvement. (*B*) Detail of first digit in *A* showing lace-like periosteal reaction of diaphysis of proximal and middle phalanges.

distinguishable with any degree of certainty from those associated with the involvement in rheumatoid arthritis and the invariable bilateral sacro-iliitis in ankylosing spondylitis. There appears to be less extensive destruction and synostosis in Reiter's syndrome than is usually seen in the sacro-iliac joints of ankylosing spondylitis.

Osseous, articular, and ligamentous changes in the spine which are characteristic findings in ankylosing spondylitis were rarely seen in this series of Reiter's syndrome, even when there was destruction of the sacro-iliac joints. This differing incidence of spinal changes is an important roentgenographic feature differentiating these two diseases. Some investigators[3,6] have suggested that classic ankylosing spondylitis may occasionally follow Reiter's syndrome. Only one case in this series would suggest this occurrence (Fig. 9). There is sufficient difference in the mode of onset, clinical pattern, skin and mucosal lesions, and incidence of spinal and peripheral involvement to separate the two as distinct entities.

Roentgenographic changes in the symphysis pubis, indistinguishable from ankylosing spondylitis, are also a recognized feature in Reiter's syndrome. Two such cases were observed in this series.

Murray *et al.*[7] noted that the roentgenographic changes in Reiter's syndrome were similar in many respects to those seen in rheumatoid arthritis and in some cases of ankylosing spondylitis. These authors emphasized that there is a limited number of ways in which a bone or joint can react to a pathogenic agent and that different etiologic factors may cause identical roentgenographic changes.

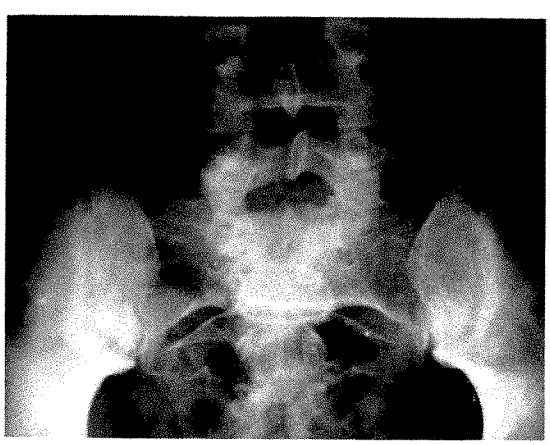


Fig. 8. Severe bilateral involvement of 18 months' duration of sacro-iliac joints.

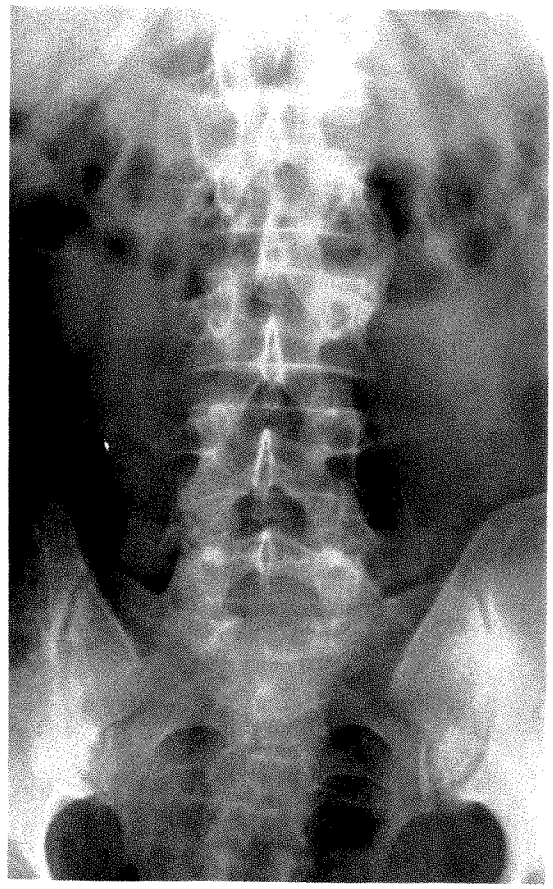


Fig. 9. Unilateral calcific bridging on left between L_2 and L_3. Similar findings were noted between T-12 and L-1 on right. Note bilateral early erosive changes in the sacro-iliac joints.

A comparison of the roentgen features of Reiter's syndrome, rheumatoid arthritis, and ankylosing spondylitis by Mason *et al.*[8] stressed that the characteristic florid periostitis in the calcaneus is the only significant differentiating factor, although minor differences in the incidence and distribution occur in other bones and joints. Most investigators have commented on the findings of periosteal new bone formation, es-

pecially in the calcaneus. Others have emphasized osteoporosis. These findings are further confirmed in our series of 46 cases.

SUMMARY AND CONCLUSIONS

The roentgenograms of 46 cases of Reiter's syndrome were reviewed to determine what roentgen features might suggest the diagnosis. The evidence found indicates that this syndrome has a consistent roentgenologic pattern. The high incidence of involvement of the bones and joints of the lower extremity and sacro-iliac is noted. Severe changes were found in patients who had been ill a prolonged period of time or in those who had repeated attacks. The similarities and differences between Reiter's syndrome and rheumatoid arthritis are described. A characteristic fluffy prolific periosteal reaction on the plantar surface of the calcaneus is believed to be specific for Reiter's syndrome.

William V. Weldon, Capt., MC
Radiology Service
Walter Reed General Hospital, WRAMC
Washington 12, D. C.

REFERENCES

1. Brodie, B. C. Pathological and Surgical Observations on Diseases of the Joints. Longmans, London, 1818. Second edition, 1836.
2. Csonka, G. W. Course of Reiter's syndrome. *Brit. M. J.*, 1958, *1*, 1088–1090.
3. Ford, D. K. Natural history of arthritis following venereal urethritis. *Ann. Rheumat. Dis.*, 1953, *12*, 177–197.
4. Grainger, R. G. Procto-colitis and other pelvic infections in relation to ankylosing spondylitis. *J. Fac. Radiologists*, 1959, *10*, 138–150.
5. Marche, J. L'atteinte des articulations sacro-iliaques dans le syndrome dit de Reiter. *Rec. rhum.*, 1950, *17*, 449–451.
6. Mason, R. M., Murray, R. S., Oates, J. K., and Young, A. C. Comparative radiological study of Reiter's disease, rheumatoid arthritis and ankylosing spondylitis. *J. Bone & Joint Surg.*, 1959, *41B*, 137–148.
7. Murray, R. S., Oates, J. K., and Young, A. C. Radiological changes in Reiter's syndrome and arthritis associated with urethritis. *J. Fac. Radiologists*, 1958, *9*, 37–43.
8. Oates, J. K. Sacro-iliitis in Reiter's disease. *Brit. J. Ven. Dis.*, 1958, *34*, 177–180.
9. Reiter, H. Über ein bisher unerkannte Spirochaetinfektion (spirochaetosis arthritica). *Deutsche med. Wchnschr.*, 1916, *42*, 1535–1536.
10. Reynolds, D. F., and Csonka, G. W. Radiological aspects of Reiter's syndrome ("venereal" arthritis). *J. Fac. Radiologists*, 1958, *9*, 44–49.

VOL. 86, NO. 2

TUMORAL CALCINOSIS*

REPORT OF THREE CASES AND REVIEW OF THE LITERATURE

By DEWEY L. BARTON, CAPTAIN, MC, USA,† *and* ROBERT J. REEVES, M.D.
DURHAM, NORTH CAROLINA

THE abnormal deposition of calcium salts in the soft tissues is termed calcinosis. Two major types have been generally accepted, namely, calcinosis universalis and calcinosis circumscripta. The former occurs primarily in children and consists of small, hard, calcified nodules and plaques in the skin, subcutaneous tissue and superficial muscle layers. The calcifications are widespread with major involvement of the extremities. Muscle atrophy, contractures, and ankylosis may occur in later stages. The prognosis is generally poor and scleroderma is present in approximately 40 per cent of the cases.

The circumscribed type is usually seen in middle-aged women and has a benign, slowly progressive course. The calcium deposits consist of small nodules and streaks, located primarily about the flexor tendon sheaths in the hands and wrists; occasionally about the elbow and anterior aspect of the knee. Raynaud's disease, scleroderma or sclerodactyly is associated in 30 to 40 per cent of the cases.

A rare pattern of calcinosis, characterized by large, nodular periarticular deposits has been described under various titles.[13,20,22,33,34] In 1943, Inclan[13] introduced the term "tumoral calcinosis." The symptoms, clinical course and roentgenologic aspects do not conform to those described in the universal and circumscribed forms, and recognition of it as a third major type of calcinosis seems justified. Only a few cases have been reported since the publication of Inclan's paper and none have been noted in the American radiologic literature. It is the purpose of this paper to present 3 cases and briefly review the literature.

REVIEW OF THE LITERATURE

Calcinosis has been observed in considerable variety, degree and distribution. Transitional forms and overlap occur, making classification of certain cases into particular types difficult or impossible. However, a majority of the reported cases can be satisfactorily classified as calcinosis universalis or circumscripta. In 1931, Steinitz published a comprehensive review which included 71 cases of calcinosis circumscripta and 34 of calcinosis universalis. In another review, Rothstein and Welt,[27] in 1936, noted 39 cases in children up to fifteen years of age. Of this group, 32 were classified as calcinosis universalis and 7 as calcinosis circumscripta. By 1952, at least 150 cases of the circumscribed form had been reported.[14]

Descriptions of the tumoral type are much less common. In 1935, Teutschlaender[34] used the term lipocalcinogranulomatosis to describe symmetric "calcium tumors" which he observed in a three year old child. He reviewed other cases in the literature which he felt represented the same entity and postulated the pathogenesis on the basis of pathologic findings. He believed that spontaneous fat necrosis was the initial event, followed by calcium deposition and granuloma formation. In his experience lipocalcinogranulomatosis is always found in young people. He regarded it as a rare variety of calcinosis universalis and suspected a hereditary factor.

Sommer and Tress,[33] in 1941, made reference to Teutschlaender and described calcium tumors and chronic renal disease in a forty-three year old female. In addi-

* From the Department of Radiology, Duke University Medical Center, Durham, North Carolina. Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

† Resident in Radiology.

tion, they mentioned the case of a seventeen year old boy with 5 large calcium deposits, reported by Hoche. Bedetti,[4] in 1946, Oosthuizen *et al.*,[20] in 1950, and Halper,[12] in 1952, reported single cases and referred to Teutschlaender.

Beginning with Inclan in 1943, a series of cases have been reported which were called "tumoral calcinosis." Inclan described the findings in 3 Negroes, aged ten, eighteen and nineteen years. They were not related. Ghormley, in a discussion following the paper, mentions a fourteen year old boy and his 2 sisters with similar calcium deposits. In 1949, Thomson and Tanner[35] added 3 more cases, all Negro male siblings, aged twelve, fifteen and twenty-five years. Palmer,[22] in 1958, reported 2 African males.

Study of the various cases leaves little doubt that Teutschlaender's lipocalcinogranulomatosis and Inclan's tumoral calcinosis are the same entity.

ROENTGENOLOGIC FINDINGS

The calcium deposits are characteristically periarticular and measure from 1 to 20, or more, cm. in diameter. They are dense, irregular and have a round or oval contour. Radiolucent fibrous septa separate clumps of calcium, giving a multinodular appearance. There are no bony abnormalities. The sites of preference are the lateral and superior aspects of the shoulder; the posterior aspect of the elbow and laterally about the hips or buttocks. Other less common sites include the lateral aspect of the feet, and the retroscapular, acromioclavicular, sacral and ischial regions. An occasional deposit has been noted in the neck, and even more rarely, in the hand and in the region of the distal femur. The deposits occupy the subcutaneous and muscle layers. Unless complicated by chronic renal disease or possibly some other abnormality, no other calcification is present. There may be bilateral, symmetric involvement.

CLINICAL FINDINGS

Appearance of the tumors is usually noted in the first or second decade. They are often asymptomatic and slowly increase in size over a period of months or years. Deposits about the hips may not be apparent until they are 8 to 10 cm. in diameter. They are fixed to the underlying tissue, and feel firm and rubbery. There is usually no tenderness, but discomfort was noted in some instances on attempt to move the masses. Occasionally, there is associated pain (Case III), which may be related to a rapid increase in size of the tumor. The overlying skin is stretched but otherwise appears normal except for sites of previous surgical drainage, or for the presence of a fistulous tract when the area has become secondarily infected. Some patients mention trauma to the area prior to the appearance of the tumor, but this history has not been common or consistent.

No limitation of movement is present in the corresponding joint except when very large masses interfere with muscle contraction. The patients have been in good health with few exceptions. One patient reported by Thomson and Tanner[35] eventually expired in a cachectic state and secondary amyloidosis was found on autopsy. Riemenschneider and Ecker[25] reported a single 4×6 cm. deposit in the left lumbosacral region of a fifty-nine year old woman. Associated symptoms of sciatica were relieved by surgical removal of the tumor.

Scleroderma has not been reported in tumoral calcinosis, in contrast to this com-

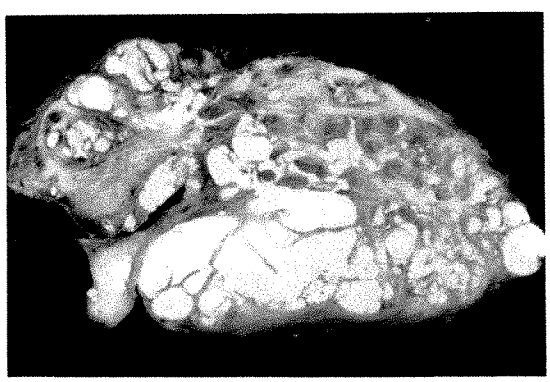


Fig. 1. Case I. Cut surface of the mass removed from the hip. Note the numerous cyst-like cavities filled with pasty, white material. Analysis showed this to be mainly calcium phosphate.

mon finding in calcinosis universalis and circumscripta. The prognosis is good. A number of the patients who have been followed for years did not develop any significant complications.

The presence of tumors in more than one member of a family is a remarkable feature in the small number of reported cases. This is very rare in calcinosis universalis and circumscripta. Laboratory findings are characteristically within normal limits, excluding the patients with chronic renal disease.

PATHOLOGIC OBSERVATIONS

The tumors are firm, rubbery and multilobular with a fibrous tissue covering. When removed, they were found to be firmly fixed by fibrous adhesions to the surrounding muscles, fascia and tendons. The nearby bursae are usually not involved. Sectioning of the tumors reveals a multilocular structure with fibrous connective tissue bands separating pasty, calcareous deposits (Fig. 1). The fibrous walls are lined with multinucleated giant cells, macrophages containing lipoid substance and inflammatory cells (Fig. 2, *A* and *B*). Cultures and stains are negative for any organisms. Analysis of the calcareous material reveals mainly calcium phosphate and has been of no particular help in distinguishing these deposits from those occurring in other forms of calcinosis.

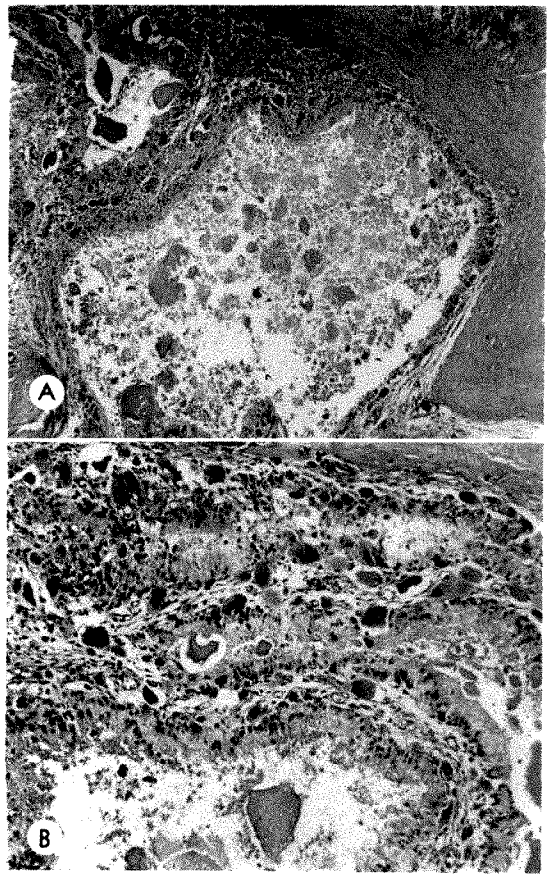


Fig. 2. Case III. (*A*) Section of mass showing large areas of calcification surrounded by dense fibrous tissue (low power magnification 71.5×). (*B*) Many multinucleated giant cells, epithelioid cells, macrophages containing lipoid substances and foamy plasma cells are present (high power magnification 124×).

DIFFERENTIAL DIAGNOSIS

The clinical and roentgenologic findings in calcinosis universalis and circumscripta have already been briefly described. The distinction between these entities and tumoral calcinosis should be possible by both roentgen and clinical features. Occasionally, a case demonstrates more than one pattern of calcinosis. Moore and Clarke[18] reported the case of a forty-six year old female with scleroderma. Roentgenologically, there were features of all three types of calcinosis.

Although metastatic calcification may occur in many diseases, massive periarticular calcification is uncommon. The only entities noted in the literature with calcium deposits similar to those in tumoral calcinosis are in hypervitaminosis D and milk drinker's syndrome. A pertinent history should be obtainable in both of these diseases, as well as the presence of an elevated serum calcium and phosphorus. In addition, other sites of metastatic calcification are usually a prominent feature. The cases of milk drinker's syndrome reported by Poppel and Zeitel[24] showed widespread vascular calcification and deposits in the falx cerebri, kidneys and lungs. In hypervitaminosis D there is usually an associated general decrease in bone density.[7]

Four cases of calcinosis described in patients with chronic renal disease, including

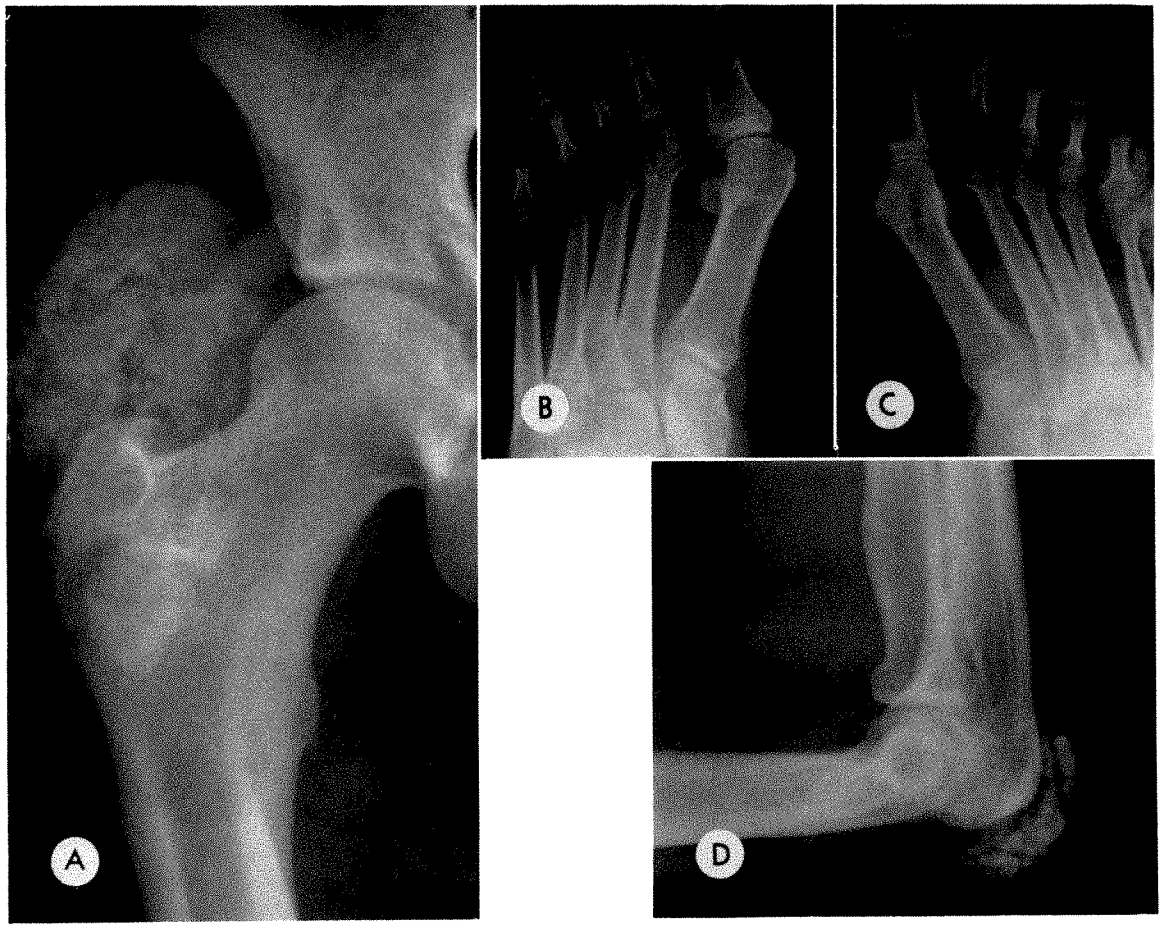


Fig. 3. Case I. (*A*) The left hip, (*B*) left foot, (*C*) right foot, and (*D*) right elbow. *C* and *D* are common sites of involvement.

Case III, presented here, showed "tumors" which are indistinguishable roentgenologically and pathologically from the cases reported as tumoral calcinosis and lipocalcinogranulomatosis.[6,9,33] One significant difference was in the late onset of the deposits in these patients in contrast to the onset in the first or second decade in the others.

REPORT OF CASES

Case I. A twenty-eight year old Negro male was first seen at Duke Hospital in 1945, at the age of thirteen years. A small calcium mass was removed opposite the first metatarsophalangeal joint on the right foot. He was next seen in January, 1960, at which time he had tumoral collections of calcium posteriorly on the right elbow, about the left hip, right lateral aspect of the foot and a 2 cm. nodule over the right acromioclavicular joint (Fig. 3, *A-D*). Physical examination revealed no other abnormalities except for the presence of angioid streaks in both retinae. He stated that the masses had developed slowly over a number of years. His general health was good and he worked regularly as a farmer. There was no limitation of joint movement and no bony abnormalities. He did have recurrent symptoms of secondary infection in the deposit about the hip and this had been drained on two occasions. All of the masses were removed surgically. Pathologically, the findings were the same as those described in previous reports of tumoral calcinosis and lipocalcinogranulomatosis (Fig. 4, *A* and *B*).

Laboratory studies showed a normal urinalysis and hemogram. Three serum calcium and phosphorus determinations were 10.4, 9.8, 10.6

and 6.3, 5.8 and 7 mg. per cent, respectively. Alkaline phosphatase was 1.6 Bodansky units.

CASE II. A thirty-two year old Negro male, brother of Case I, was examined when it was learned that he had similar subcutaneous masses removed from the right hip, both elbows and the right foot at the age of twelve years. He had been asymptomatic since that time and was not aware of any recurrence. A roentgen survey did not reveal any deposits at the sites of previous surgery but a 5×9 cm. collection of calcium was present about the left hip (Fig. 5).

Physical examination revealed angioid streaks in the retina, similar to those of his brother, but no other abnormalities. The tumor about the left hip was sufficiently deep to be overlooked. The serum phosphorus was slightly elevated, 5.5 mg. per cent. The serum calcium was 8.5 mg. per cent and other laboratory studies were negative.

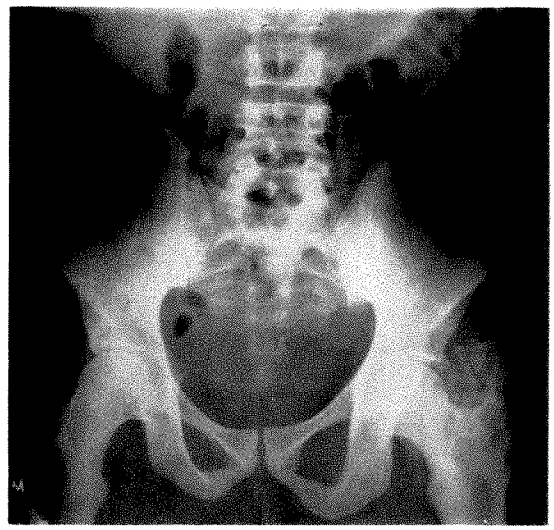


FIG. 5. Case II. Roentgenogram demonstrates the asymptomatic calcium deposit. A history of similar deposits about the right hip, both elbows and right foot, removed twenty years previously, was presented.

The father, mother, a sister and another brother were examined and no abnormalities were found.

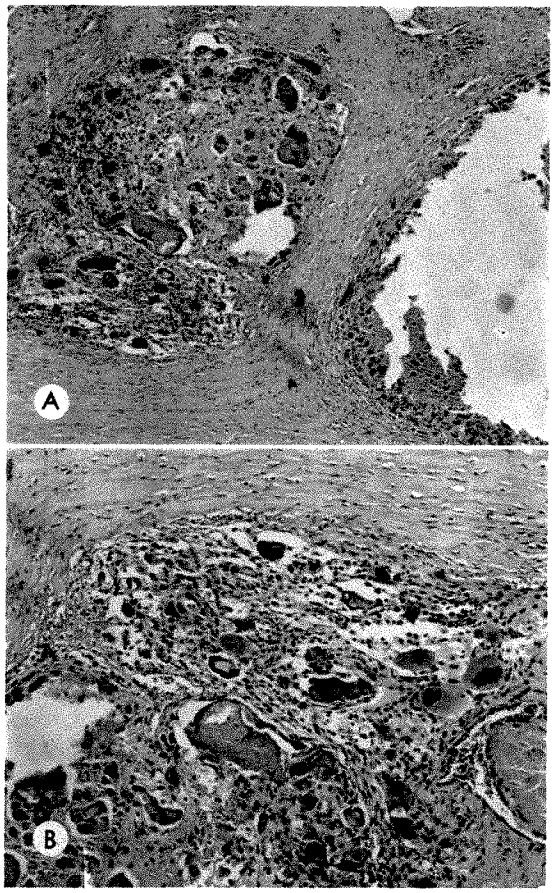


FIG. 4. (*A*) Case I. Section of mass shows dense fibrous bands separating the calcium deposits (low power magnification 71.5×). (*B*) Same cell types as noted in Case III. No bacteria, fungi or other organisms were seen (high power magnification 124×).

CASE III. A thirty-two year old thin white female was admitted to Duke Hospital in September, 1957, with a one year history of enlarging subcutaneous masses about both shoulders, elbows, hips and feet (Fig. 6, *A–F*). Roentgenograms revealed these to be dense, multilobular deposits of calcium without any bony abnormalities.

She complained of general malaise, anorexia and a 30 pound weight loss. Periodically, she experienced pain in the region of the masses during the six months prior to admission, when the growth was especially rapid. The overlying skin was stretched, but otherwise appeared normal. Physical examination revealed metastatic calcification in the conjunctiva, cornea, and tympanic membranes bilaterally. In addition, there was bilateral renal calcification and faint vascular calcification in the pelvis.

Excretory urograms showed poor function and phenolsulfonphthalein excretion was 20 per cent in two hours. Serum calcium ranged be-

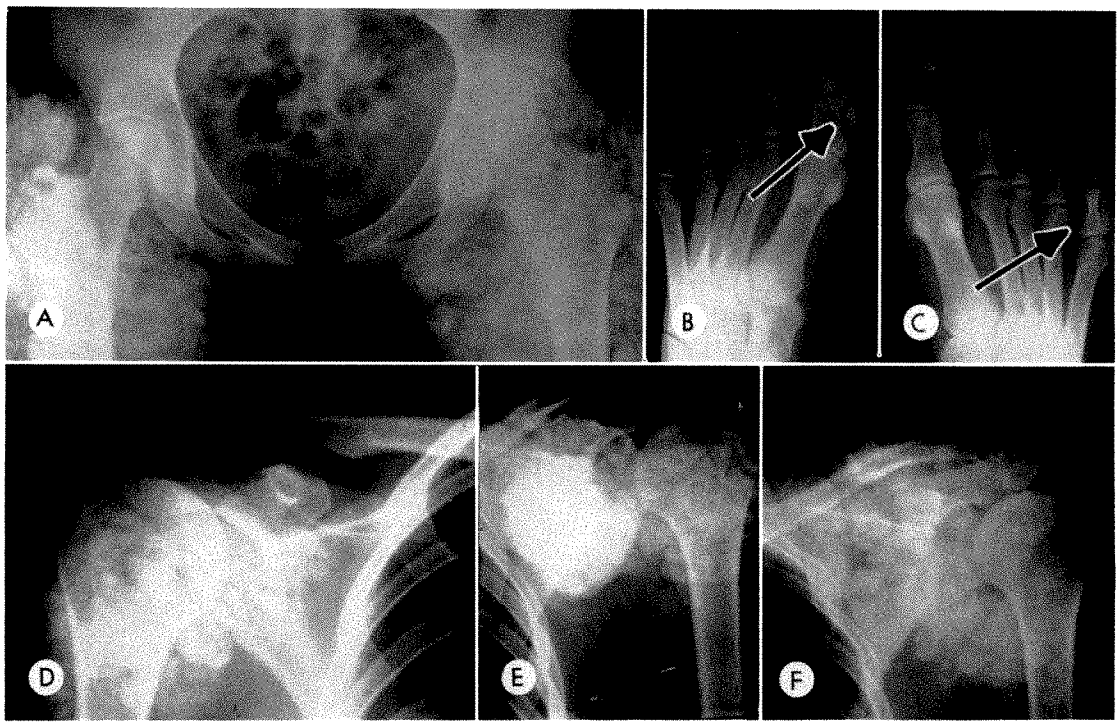


FIG. 6. Case III. Calcium deposits about both hips (*A*), feet (*B* and *C*), and shoulders (*D* and *E*). The massive deposits about the hips extend into the upper thighs. Note in *F*, taken two years after *E*, the increase in deposition about the left shoulder.

tween 10 and 12.6 mg. per cent; serum phosphorus between 4 and 5 mg. per cent. Nonprotein nitrogen was 61 mg. per cent.

Treatment with cortisone and a low calcium and phosphorus diet was instituted but the calcium deposits remained unaltered. On a second admission in March, 1959, a parathyroid exploration was performed. The glands appeared normal at surgery but three were removed. Sections of one gland showed a zone of fibrosis with numerous small tubercles and several multinucleated giant cells. This raised the question of sarcoidosis, but no additional supportive evidence was obtained. A liver biopsy was negative. The serum calcium and phosphorus levels did not change postoperatively.

A portion of the large mass about the left hip was removed in November, 1959. The pathologic findings were the same as those in Case I, (Fig. 2, *A* and *B*).

DISCUSSION

The first 2 cases presented here show the clinical features described in previous reports, namely, an onset in the first or second decade, familial tendency and a benign clinical course. The large mass in the left hip of the younger brother (Case I) became secondarily infected on a few occasions but otherwise the deposits caused no particular discomfort and did not interfere with activity. In a few of the other cases reported, this was also a complication. Case I and II had slightly elevated serum phosphorus levels without apparent cause. This has not been previously reported.

The presence of angioid streaks in the retina in both Case I and II aroused additional interest. Angioid streaks are most commonly associated with pseudoxanthoma elasticum;[28] however, they have also been reported in people with Paget's disease, and recently in 2 cases of sickle cell anemia.[13] The remainder of cases presenting angioid streaks is a small miscellaneous group, including some in which these were reported as incidental findings. Neither Case I nor Case II had observable skin changes and biopsies were negative. It is unlikely that pseudoxanthoma elasticum has any relationship to tumoral

calcinosis. Although vascular calcification is common in pseudoxanthoma elasticum, only one case of the hundreds reported showed soft tissue calcium deposits. This was in a sixty-nine year old white male who had a diffuse deposit of calcium plaques in the soft tissues of the right leg.

The roentgenologic and pathologic findings of the calcium tumors were the same in all 3 cases, but the late occurrence of the tumors and the presence of renal insufficiency with metastatic calcification in Case III are distinctive features. Three other cases have been found in the literature in which tumoral deposits of calcium appeared in people with chronic renal disease.[6,9,33] In these cases the appearance of the tumors was in the fifth decade or later and only after development of renal insufficiency. Although metastatic calcification is common in renal insufficiency of long standing, large periarticular masses of calcium are definitely rare. The relationship between these cases and the younger group who are in good health is not clear.

The pathogenesis of the calcium tumors has not been established. The presence of foamy plasma cells and lipoid substances in the macrophages has attracted most attention, suggesting some origin from fatty tissue. Teutschlaender[34] proposed that the process begins with spontaneous necrosis of fat and this is followed by granuloma formation and calcification. A relationship to the bursae has been considered but in most cases they were not involved. Also, some masses have occurred where there are no bursae.

The only satisfactory therapy has been surgical removal of the masses. The earlier in development that they are removed, the easier and more complete the removal. Although new masses may appear, there is usually no recurrence at the sites of removal and the prognosis appears to be very good.

SUMMARY

Three cases of calcinosis characterized by large, nodular, periarticular deposits are presented. The first 2 cases are Negro siblings who show the typical clinical features, namely, onset in the first or second decade, familial tendency, and a benign clinical course. The third case is an example of calcium tumors in association with chronic renal disease.

This form of calcinosis was described by Teutschlaender[34] in 1935 as lipocalcinogranulomatosis and in 1943 by Inclan[13] as tumoral calcinosis. Because of the use of various terms, the actual incidence is difficult to determine but it appears to be more rare than calcinosis universalis or circumscripta. The symptoms, course and roentgen aspects do not conform to those described in the universal and circumscribed forms and recognition of it as a third major type seems justified.

Dense, nodular masses are seen on roentgenograms, primarily about the hips, shoulders and elbows. Smaller areas may occur adjacent to the spine and about the wrists, feet, lower ribs, and sacrum.

The tumors have a multilocular structure with fibrous connective tissue bands separating pasty, calcareous deposits. Microscopically, macrophages, multinucleated giant cells and chronic inflammatory cells lining the fibrous bands are noted.

Laboratory studies are usually within normal limits and the etiology is obscure. The only effective therapy has been surgical removal of the masses. The prognosis is good. A number of patients who have been followed for years have not developed any significant complications or limitation of activity. The literature is briefly reviewed.

Robert J. Reeves, M.D.
Department of Radiology
Duke University Medical Center
Durham, North Carolina

REFERENCES

1. Albright, F., and Reifenstein, E. C., Jr. Parathyroid Glands and Metabolic Bone Disease. Williams & Wilkins Company, Baltimore, 1948.
2. Barr, D. P. Pathological calcification. *Physiol. Rev.*, 1932, *12*, 593–624.
3. Bauer, W., Marble, A., and Bennett, G. A. Further studies in case of calcification of subcutaneous tissue in child. *Am. J. M. Sc.*, 1931, *182*, 237–251.

4. Bedetti, D. Sulla cosidetta (lipocalcinogranulomatosi delle borse mucose) e suoi rapporti colla calcinosi interstiziale giovanile. *Radiologica*, Rome, 1946, 2/4, 223–248.
5. Bolam, M. Calcinosis; with report of case of calcinosis universalis. *Brit. J. Dermat.*, 1935, *47*, 340–351.
6. Burkholder, T. M., and Braund, R. R. Massive calcinosis with chronic renal insufficiency due to polycystic kidneys; case report. *J. Urol.*, 1947, *57*, 1001–1009.
7. Cardoen, G. Le syndrome "calcinose." *Acta chir. belg.*, 1951, *50*, 545–562.
8. Christensen, W. R., Liebman, C., and Sosman, M. C. Skeletal and periarticular manifestations of hypervitaminosis D. Am. J. Roentgenol. & Rad. Therapy, 1951, *65*, 27–41.
9. Comroe, B. I., Chamberlin, G. W., and Sunderman, F. W. Interstitial calcinosis; report of case and review of literature. Am. J. Roentgenol. & Rad. Therapy, 1939, *41*, 749–757.
10. Curtis, L. E., and Feller, A. E. Hyperparathyroidism with calcinosis and secondary to renal disease. *Ann. Int. Med.*, 1942, *17*, 1005–1014
11. Epstein, E. Idiopathic calcinosis cutis; review, with report of two cases. *Arch. Dermat. & Syph.*, 1936, *34*, 367–377.
12. Halper, H. Calcinosis with description of case of calcinosis circumscripta. *Brit. J. Radiol.*, 1952, *25*, 584–588.
13. Inclan, A. Tumoral calcinosis. *J.A.M.A.*, 1943, *121*, 490–495.
14. Ingram, M. D., Jr. Calcinosis in scleroderma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1952, *68*, 918–921.
15. Jaffe, H. L. Primary and secondary (renal) hyperparathyroidism. *S. Clin. North America*, 1942, *22*, 621–639.
16. Levin, R. T., and Genovese, P. D. Report of case of long standing renal insufficiency with extensive metastatic calcifications. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 423–429.
17. Longcope, W. T., and Freiman, D. G. Study of sacroidosis. *Medicine*, 1952, *31*, 1–132.
18. Moore, M., Jr., and Clarke, C. L. Extreme calcinosis interstitialis; report of case. *South. M. J.*, 1950, *43*, 861–863.
19. Mulligan, R. M. Metastatic calcification. *Arch. Path.*, 1947, *43*, 177–230.
20. Oosthuizen, S. F., Le Roux, P., and De Wet, A. J. Calcino-universalis: type lipo-calcinogranulomatosis. *Brit. J. Radiol.*, 1950, *23*, 598–600.
21. Orton, G. H. Calcium changes and their importance in diagnostic radiology. *Brit. J. Radiol.*, 1936, *9*, 102–125.
22. Palmer, P. E. S. Massive (or tumoural) calcinosis. *Brit. J. Radiol.*, 1958, *31*, 104–105.
23. Paton, D. Angioid streaks and sickle cell anemia; report of two cases. *Arch. Ophth.*, 1959, *62*, 852–858.
24. Poppel, M. H., and Zeitel, B. E. Roentgen manifestations of milk drinker's syndrome. *Radiology*, 1956, *67*, 195–199.
25. Riemenschneider, P. A., and Ecker, A. Sciatica caused by tumoral calcinosis. *J. Neurosurg.*, 1952, *9*, 304–307.
26. Rosenberg, E. F. Chalk gout. *J.A.M.A.*, 1940, *115*, 1791–1794.
27. Rothstein, J. L., and West, S. Calcinosis universalis and calcinosis circumscripta in infancy and in childhood; 3 cases of calcinosis universalis, with review of literature. *Am. J. Dis. Child.*, 1936, *52*, 368–422.
28. Scholz, R. O. Angioid streaks. *Arch. Ophth.*, 1941, *26*, 677–695.
29. Scholz, T. Diffuse interstitial calcinosis; report of case, with review of literature. *Radiology*, 1934, *22*, 54–66.
30. Shaffer, B., Copelan, H., and Beerman, H. Pseudoxanthoma elasticum. *Arch. Dermat.*, 1957, *76*, 622–633.
31. Snapper, I. Bone Diseases in Medical Practice. Grune & Stratton, New York, 1957.
32. Soeur, R. La calcinose. *Acta chir. belg.*, 1950, *49*, 240–248.
33. Sommer, F., and Tress, E. Beitrag zum Krankheitsbilde der Lipokalzinogranulomatose (eine besondere Form der Calcinosis universalis). *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1941, *63*, 205–214.
34. Teutschlaender. Über progressive Lipogranulomatose der Muskulatur. Zugleich ein Beitrag zur Pathogenese der Myopathia osteoplastica progressiva. *Klin. Wchnschr.*, 1935, *14*, 451–453.
35. Thomson, J. E. M., and Tanner, F. J. Tumoral calcinosis. *J. Bone & Joint Surg.*, 1949, *31-A*, 132–140.

VOL. 86, No. 2

ARTHROGRAPHY OF THE KNEE*

A CRITICAL STUDY OF ERRORS AND THEIR SOURCES

By I. KESSLER, M.D., Z. SILBERMAN, M.D., *and* F. NISSIM, M.D.
TEL-AVIV, ISRAEL

MANY papers dealing with arthrography of the knee have already been published. Lindblom[5] in 1948 described his extensive experience in arthrographic procedures and stated the basis for the interpretation of findings as well as his percentage of errors. Various reports have appeared since then. We do not intend to present here another discussion on the principles of arthrographic interpretation; our purpose is merely to analyze this method as a means of diagnosing semilunar cartilage injuries.

The following considerations led to the preparation of this paper: (1) On reviewing the literature, we were impressed by the great differences in arthrographic interpretation by different authors. Whereas the incidence of errors reported by some is as low as 5 per cent (Lindblom[5]), it ranges up to 87.5 per cent in other reports (Oggioni[7]). Obviously, as long as the rate of error does not exceed 5 per cent, this procedure can be considered as an essential aid in the diagnosis of knee lesions. However, errors exceeding this percentage, particularly if they reach as high as 87.5 per cent, will definitely lower its value to a point which does not justify its use. (2) After the great initial enthusiasm for this procedure, there is now a tendency in many clinics to refrain from performing arthrography and, consequently, treatment of knee lesions, including surgical procedures, is performed without its use.

We believe that the situation now requires a thorough re-evaluation of the subject in order to objectively approve or reject the use of arthrography of the knee.

MATERIAL AND METHOD

Since 1957 we have performed 350 arthrographies of the knee using a contrast solution. After a thorough study of the different arthrographic techniques, the positive contrast medium method was accepted in our clinic because the shadow obtained by using air or oxygen was dim in comparison with the sharpness produced by opaque contrast solutions. In addition to this advantage, except for slight swelling in a few cases during the first twenty-four hours, no secondary ill-effects are mentioned in the literature with the use of opaque solutions, whereas air and oxygen are known to cause embolism and synovial reactions. Another complication was reported in 1950 by Murray and Forrai,[6] who observed the appearance of eosinophilia in the synovia and synovial fluid after pneumoarthrography. In spite of these disadvantages, gas is still used as a contrast medium in different clinics (Bonnin and Boldero,[1] and Keats *et al.*[3]). Without dwelling upon further details, and by no means intending to discount the value of a negative contrast medium, we are considering in this report arthrography with opaque chemical solutions only. In all our cases a 35 per cent solution of diodone B.P. was injected. The volume of the solution introduced varied between 8 and 12 cc., depending on the size of the knee.

The investigations are divided into two groups. The first group comprises 150 arthrographies performed using the usual technique; in the second group of 200 studies, some new modifications in technique were introduced. The arthrographies were performed on patients with various complaints in their knees, usually after trauma. In 46 per cent (160 instances) lesions of the semilunar cartilage were more or less evident from the clinical signs. In all the remaining cases the clinical diagnosis was not clear. The age of the patients

* From the Department of Traumatology and the Department of Orthopaedic Surgery, Central Clinic "Zamenhof," Tel-Aviv, Israel.

TABLE I

RELATIONSHIP BETWEEN ARTHROGRAPHIC FINDINGS AND ERRORS

	Series I	Series II	Total
Arthrographic Examinations	150	200	350
Arthrographically Diagnosed Lesions	27	93	120
Operations	24	72	96
Errors	5	1	6

ranged from fourteen to sixty years. Twelve were females. Ninety-six patients were operated, and the arthrographic findings were confirmed in 90 instances. The 6 cases with false findings included 5 in our first group: in 3 with negative arthrograms, a tear of the semilunar cartilage was found, and in 2 the positive arthrographic findings were disproved. In the 1 case with false negative findings in our second group, a tear of the medial semilunar cartilage was found (Table 1).

We have elaborated on these statistics because they clearly justify the procedures upon which our report is based.

TECHNIQUE

The ordinary technique of injecting a contrast medium into the knee uses the lateral approach above the patellar retinaculum. By using this route the material may penetrate either the upper portion of the joint space or the lower portion of the suprapatellar bursa, which communicate freely with each other and which therefore makes this site more suitable for aspiration than injection. Sometimes, after injection of the material, even when there is no feeling of resistance, the contrast medium does not appear in the joint on the roentgenograms but shows as an irregular patch at the site of the injection. This phenomenon is explained by the fact that the solution may be introduced into the fat body which is located between the bursa and the femur. The result is complete failure of the arthrographic examination in addition to a severe, painful reaction in the knee due to aseptic inflammation of the fat body, which may last for a few days.

In our second series we preferred to use, as an injection site, the medial aspect of the joint, about 3 cm. medial to the patellar ligament when the knee is flexed. At this point the joint space is quite near the skin. The needle penetrates it immediately after puncturing the superficial layers, and the solution is introduced easily and accurately (Fig. 1, *A* and *B*). Flexing the knee subjects the joint to tension, which causes some synovial fluid to leak out through the needle, especially after injecting small amounts of procaine. This is considered to be an indication that the needle is within the joint space. There is only one warning concerning the use of this site: the puncture should be performed at least 3 cm. medial to the patellar liga-

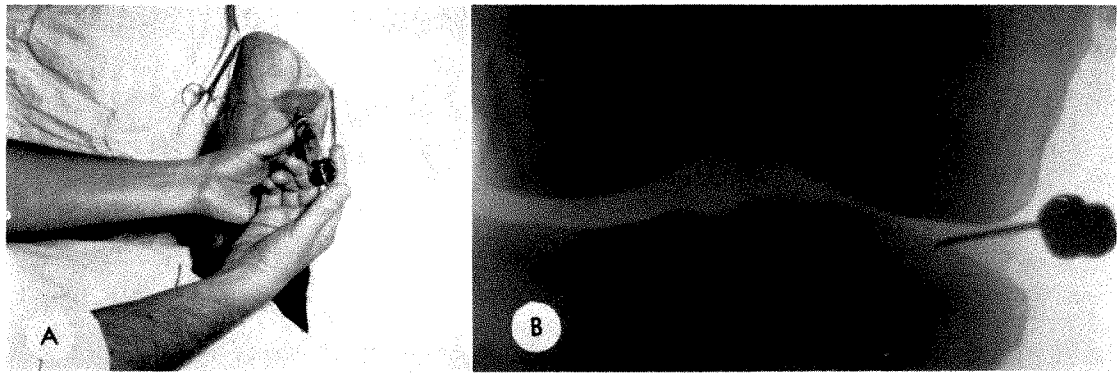


FIG. 1. (*A*) Site of the contrast medium injection at the medial aspect of the joint (when the knee is flexed) about 3 cm. medial to the patellar ligament and (*B*) arthrogram showing position of the needle.

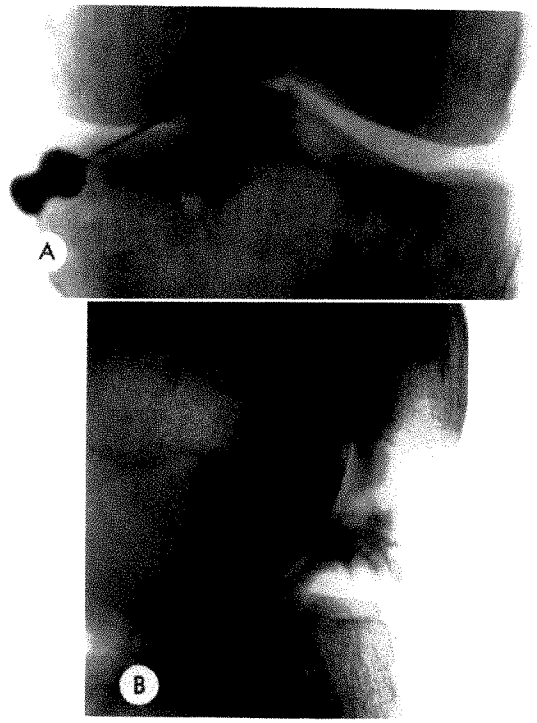


Fig. 2. (*A*) When the puncture is performed near the patellar ligament, the needle penetrates the synovial fold and (*B*) the solution is injected into the fold, outside the joint space.

ment, otherwise the needle may penetrate the synovial fold, and the solution will consequently be injected outside the joint space (Fig. 2, *A* and *B*).

If the needle is not held steady during the injection, a very small amount of the contrast material may be injected outside of the joint space (para-articular). In this case the artifact may give the impression of a false lesion of the semilunar cartilage, or it may hide an actual one. Obviously, this is one of the reasons for faulty interpretations (Fig. 3, *A* and *B*).

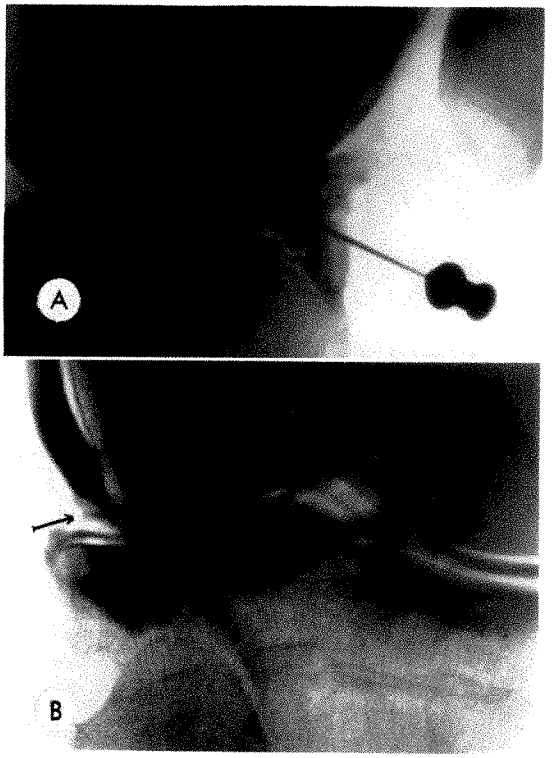


Fig. 3. (*A*) When only a small amount of contrast material is introduced para-articularly, (*B*) in external rotation the artifact (arrow) may give the impression of a lesion of the external semilunar cartilage.

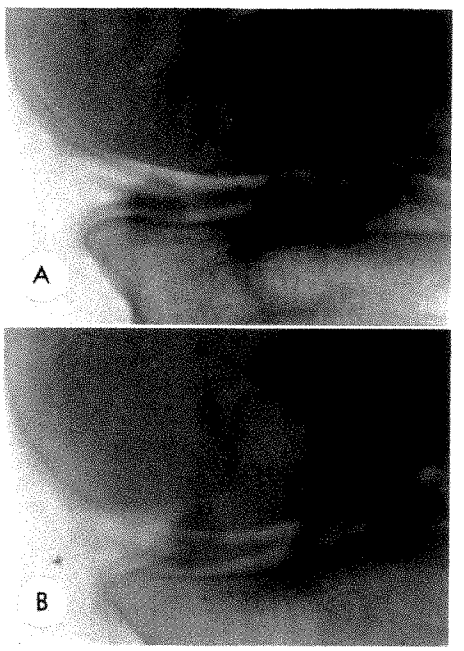


Fig. 4. (*A*) Arthrogram made immediately after injection of the contrast medium shows a tear in the posterior horn of the medial semilunar cartilage. (*B*) Ten minutes later the lesion is not clearly seen.

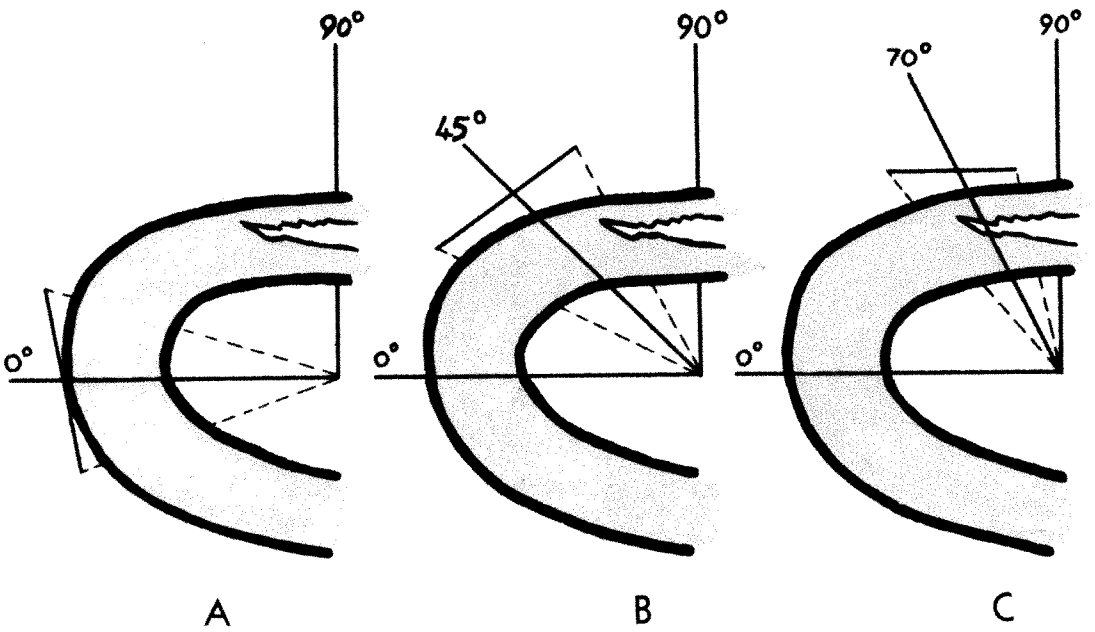


Fig. 5. (*A*, *B* and *C*) Drawings showing the value of increased rotation to reveal lesions in the vicinity of the insertion of the horn. It is obvious that only rotation of 70° will reveal this site (*C*), which will not be shown by the usual rotation of 45° (*B*).

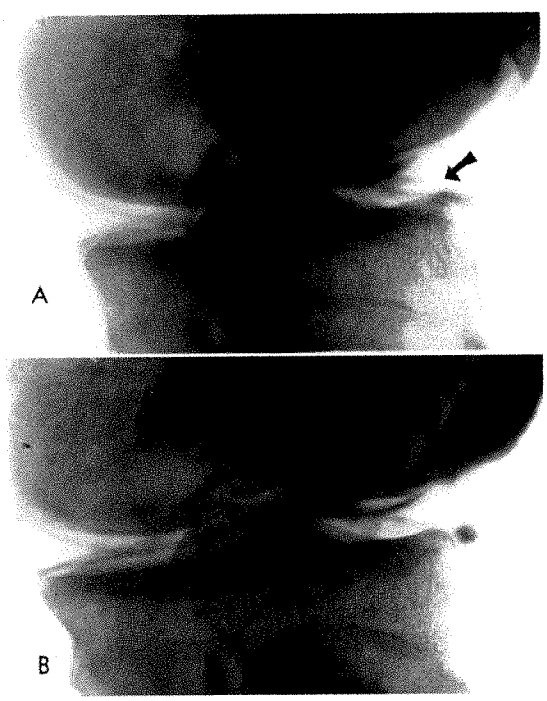


Fig. 6. D.S.95B. (*A*) A lesion (arrow) in the anterior horn of the external semilunar cartilage is demonstrable by 70° rotation. (*B*) It cannot be seen in the usual 45° rotation.

Difficulty in interpretating the arthrograms may be due to the length of time required to perform the whole procedure. In some instances the contrast medium disappears in about half an hour. In others, only dispersed traces of shadow can be seen. For this reason the procedure should take a minimum of time. Any delay will distort the contrast outlines, rendering interpretation difficult (Fig. 4, *A* and *B*.)

Some of the recesses and bursae may cause unusual contrast lines when the knee is examined in different positions. These findings have already been described in many reports and, by studying the interpretation of normal arthrograms, false findings can be avoided.

As is well known, every rotation of the knee reveals only a certain part of the semilunar cartilage. For instance, outward rotation of the knee shows the anterior horn of the lateral semilunar cartilage and the posterior horn of the medial. By increasing the rotation, other areas of the cartilage can be seen. In our opinion, the

usual rotation of 45° is not sufficient to reveal all the possible lesions in the horns. When the lesion is small and is situated in the vicinity of the horn insertion, it can be seen only by increased rotation (Fig. 5, *A*, *B* and *C*). As a standard feature of our procedure, we use a rotation of about 70°; only by this means was it possible to detect an additional number of lesions near the insertion of the horns (Fig. 6, *A* and *B*; and 7, *A*, *B* and *C*).

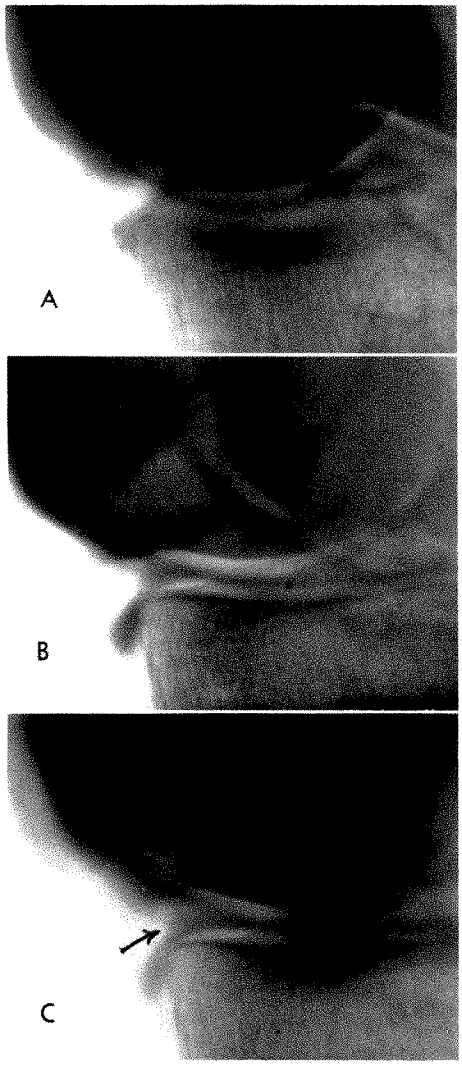


FIG. 7. K.S.73B. Internal rotation of the knee reveals the anterior horn of the medial semilunar cartilage. (*A*) Usual rotation (45°) shows a normal anterior horn. (*B*) With increased rotation (60°) a lesion is suspected. (*C*) With 70° rotation clear penetration of the contrast medium into the tear is revealed (arrow).

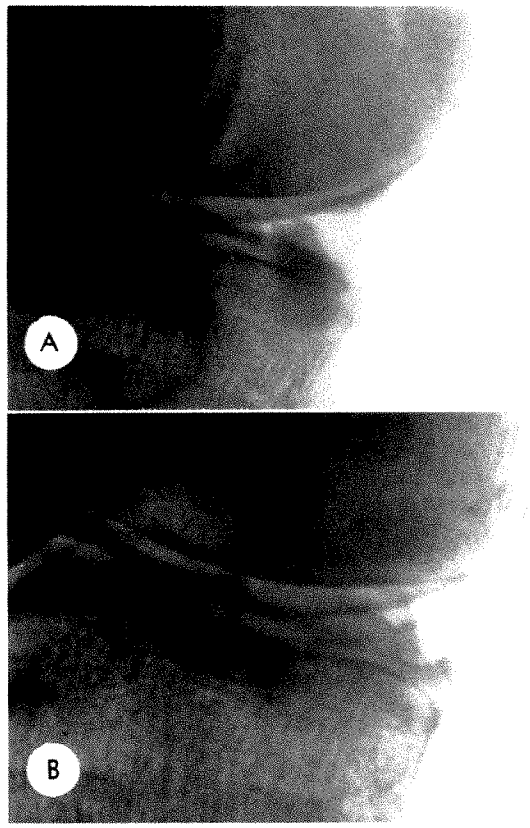


FIG. 8. A.N.65B. (*A*) Cranial projection of 10° reveals a lesion of the posterior horn of the medial semilunar cartilage. (*B*) This cannot be seen in the usual projection.

It is a well-known fact that there are partial lesions which involve the upper or the lower surface of the cartilage without penetrating the whole thickness. In such cases the contrast outlines do not appear smooth but are irregularly thickened at the site of the lesion. Frequently, these thickenings do not appear when the usual projections are used, because parts of the outline are covered by the articular surfaces of the bones. In order to eliminate this factor, a 10°–15° cranial projection has been added, which enlarges the joint space and separates the contrast outlines from the joint surfaces of the bones (Fig. 8, *A* and *B*). It should be remembered that the angle of projection should be carefully selected,

TABLE II

SUMMARY OF FINDINGS BY USUAL AND MODIFIED TECHNIQUES

Lesions	No. of Cases	Per Cent
Diagnosed by Usual Technique	66	70.9
Diagnosed by Maximal (70°) Rotation	17	18.2
Diagnosed by Craniad Projection	10	10.7
Total	93	

otherwise the bones will be superimposed in the opposite direction and the joint space will subsequently be narrowed instead of widened. Lately, laminagraphy has been used to reveal similar lesions (Klami and Kurkipää[4]). This method is rather complicated for routine use, and it is too early yet to evaluate its ultimate results.

RESULTS

As has already been mentioned, the modifications just described were applied in our second series of 200 cases. The results were as follows: of 93 arthrographically visualized lesions, 66 (70.9 per cent) were diagnosed by using the usual, standard technique, 17 (18.2 per cent) were diagnosed only by increasing the rotation, and 10 (10.7 per cent) by adding the craniad projection; in other words, 27 of 93 lesions were not diagnosed by using the accepted technique. This explains 29 per cent of possible errors (Table II). If we add to this the possibility of errors by extra-articular infiltration of the solution, quick absorption, and further unexplained causes, the percentage may reach the figures reported by different authors, as mentioned in the beginning of this paper.

CONCLUSIONS AND SUMMARY

We have not elaborated on the different features of the semilunar cartilages in respect to their anatomic variations as they appear in arthrograms of the knee. This too can be a further cause of faulty interpretation, which can be eliminated by knowing how to correctly evaluate normal arthrograms. This particular phase of the subject has been discussed by different authors.[2,5,8] The purpose of the present report is to analyze knee joint arthrography by describing the different sources of error caused by faulty performance of the procedure and by the lack of sufficient, different projections and positions.

The following points should be observed in order to obtain the best results for evaluation of arthrograms of the knee:

1. Accurate intra-articular injection of the contrast medium is the main factor for successful examination and correct interpretation. A drop of the solution injected extra-articularly, sometimes even when the needle is being extracted, can be a cause of error.

2. Lesions near the insertion of the semilunar cartilage cannot be revealed in the conventional positions. In order to visualize the site of the insertion of the horns, a further rotation of the knee to about 70° should be done.

3. Widening the visualized area of the joint space by using a further cranial projection of 10°–15° is of value in revealing incomplete and superficial lesions of the cartilage. We do not use traction of the leg when performing arthrography, because this does not always result in widening of the space and merely complicates the procedure.

4. Although we mentioned the relationship between the time used in performing the examination and accurate results, we did not elaborate on this particular point, which is a subject for investigation by itself. Our impression is that there are individual variations in the absorption of the contrast medium.

I. Kessler, M.D.
9 Aharonovitz Str.
Tel-Aviv, Israel

REFERENCES

1. Bonnin, J. G., and Boldero, J. L. Air arthrography of knee joint. *Surg., Gynec. & Obst.*, 1947, *85*, 64–70.
2. Ficat, P. L'Arthrographie Opaque du Genou. Masson & Cie, Paris, 1957.
3. Keats, T. E., Staatz, D. S., and Bailey, R. W. Pneumoarthrography of knee. *Surg., Gynec. & Obst.*, 1952, *94*, 361–364.
4. Klami, P., and Kurkipää, M. Tomoarthrography of meniscal lesions of knee joint; fifteen verified cases. *Acta radiol.*, 1957, *48*, 248–256.
5. Lindblom, K. Arthrography of knee. *Acta radiol.*, 1948, Suppl. 74.
6. Murray, R. C., and Forrai, E. Transitory eosinophilia localised in knee joint after pneumoarthrography. *J. Bone & Joint Surg.*, 1950, *32*, 74–83.
7. Oggioni, G. Cited by Klami and Kurkipää.[4]
8. Schinz, H. R., Baensch, W. E., Friedl, E., and Uehlinger, E. Lehrbuch der Röntgendiagnostik. Georg Thieme, Stuttgart, 1952.

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Published by: CHARLES C THOMAS, PUBLISHER, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued monthly. Annual subscription: United States, Mexico, Cuba, Central and South America, $15.00; Canada, $16.00; other countries, $17.00. Current single numbers, $1.50. Advertising rates submitted on application. Editorial office, 110 Professional Building, Detroit 1, Michigan, Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois. Information of interest to authors and readers will be found on page ii.

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Forty-fourth Annual Meeting: Waldorf-Astoria Hotel, New York, N. Y., April 1–4, 1962.

* *Ex officio.*

EDITORIALS

INSTRUCTION COURSES OF THE SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

PRESIDENT-ELECT Traian Leucutia has requested that we continue presenting the Instruction Courses in connection with the Annual Meeting of the American Roentgen Ray Society to be held in Miami Beach in 1961. These courses originated in 1939 during Dr. Edward Jenkinson's term as President and each succeeding president has recognized the great teaching value of this type of program and has requested its continuance.

It was felt by Dr. B. R. Kirklin, the first director, and by Dr. Harry Weber, his successor, that these courses should be conducted insofar as possible in a relatively informal manner, preferably in small groups. In order to accomplish this, it is necessary to have a large number of courses available. This plan seems like a good one and consequently the program this year will follow the same general plan of the past twenty-one years.

The schedule of the entire program, including brief abstracts of each course, the faculty list, and registration blanks appear in this issue of the JOURNAL. For your convenience the schedule of courses is printed in your program booklet also. It is possible and highly desirable to register in advance for these courses. You are advised to study the schedule of courses, select your first, second and third choice for each day and promptly mail your request to the Director. Since most of the rooms are rather small, many of the courses may be completely filled by the time of the meeting. It will be possible, however, to register at the meeting for any course not already filled. The registration area for these courses will be located in the same section of the hotel as the general registration for the entire meeting. This booth will be staffed Monday afternoon, September 25, and from 8:30 A.M. to 4:30 P.M. Tuesday through Friday, September 29.

The usual division of courses between diagnosis and therapy will obtain. Approximately two-thirds of the periods will deal with diagnostic subjects and one-third will be devoted to radiation therapy, isotopes and physics. There will be many entirely new courses added, including several with particular emphasis on special procedures, such as the one having to do with the management of cardiovascular emergencies. This is designed to alert the radiologists concerning the newest problems, which might arise and must be met in the Department of Radiology. Another extremely important course, given by the Chairmen of three leading Committees and the Chairman of the Board of Chancellors of the American College of Radiology, and moderated by Dr. Earl Barth, its immediate Past President, will deal with medical ethics and economics, radiologic organizations, and various other facts and figures which should be of great interest especially to the younger radiologists. A total of sixty-eight courses will be available, and there is a wide variation of subject matter which should prove stimulating and profitable for everyone.

Most of the courses will be held in rooms on the fourth floor of the Deauville Hotel and three rooms will be available on the mezzanine. All of the rooms are comfortably air conditioned. The courses will start at 3:00 P.M. on Tuesday, Wednesday and

Thursday and at 1:30 P.M. on Friday. Course 305, given by the officers of the American College of Radiology, will start at 1:30 P.M. on Thursday and will be held in Napoleon Room No. 3. *No tickets* will be required for this course.

We feel assured that the educational value of these courses will continue on a very high level and are very grateful to the many instructors who give so willingly of their time and talent.

HAROLD O. PETERSON, M.D.
Director, Section on Instruction

Vol. 86, No. 2

THE FORTY-THIRD ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE American Radium Society held its Forty-third Annual Meeting at the foothills of the Rocky Mountains in Colorado Springs on May 11, 12 and 13, 1961 at the Broadmoor Hotel. The attendance of members and guests eclipsed the previous meeting with a total registration of 502, representing 152 members, 216 non-member guests and 134 ladies. On Wednesday evening, preceding the official commencement of the meeting, the early arrivals were invited to participate in a Western Chuckwagon supper at the famous Flying W Ranch about twenty miles north of Colorado Springs. Three bus-loads of members attended this unique outing in the nearby mountainous ranch country for a delicious barbecued supper and western style musical entertainment with songs from "Wagon Wheels" to "Mountain Dew" by the Wranglers.

The meeting started officially on May 11 in the new parabolically constructed spacious International Center Auditorium. Dr. Juan A. del Regato, Chairman of the Arrangements Committee, welcomed the Society to its first meeting in Colorado Springs. Dr. Jesshill Love delivered the Presidential Address using for his theme the establishment of a centralized radiation center for the treatment of cancer in the smaller communities. He described the organization and facilities of the Memorial Cancer Foundation in Santa Barbara, California, which serves a radius of approximately twenty miles. Emphasis was placed on the importance of engaging a full time radiotherapist for management of the patients and directing the administration while at the same time retaining the principles of free enterprise under a contract with the Board of Trustees of the Foundation and approved by a medical board. All modern facilities are present in this center including supervoltage therapy and an isotope laboratory equipped to perform all useful procedures. Dr. Love hoped that this endowed Foundation in his community would be helpful in serving as a model which might prove instructive and encouraging to other smaller communities.

The first scientific session under the Chairmanship of Dr. Robert L. Brown consisted of presentations on the concept of a cure by Dr. Fred C. D. Collier of Birmingham and a comparative study of supervoltage radiotherapy techniques in some pelvic malignant tumors by Dr. Jose Noriega of the National Cancer Institute of Mexico. A symposium moderated by Dr. Philip Rubin on a critical analysis of the current therapy of carcinoma of the ovary was presented by 6 essayists in an attempt to answer the following questions: (1) What is the natural history of ovarian neoplasms? (2) Is surgery alone adequate? (3) Has postoperative irradiation proven itself? (4) Is preservation of the uterus worthwhile? (5) Of what value are prophylactic isotopes? (6) What is the promise of chemotherapy? The latter part of the morning session was devoted to papers on observations of radioresistant neoplasms and the influence of the time factor on the dose response curve.

A luncheon meeting of members, guests and ladies was held in the International Center Dining Room during which Dr. E. Dale Trout of the General Electric Company delivered an interesting historical talk on the life and work of T. Christen who was the first scientist to use the half-value layer as a means of specifying radiation beam quality.

The afternoon session began with the presentation of experimental work prepared by Dr. Lacassagne of the Institut du Radium of Paris. Because of Dr. Lacassagne's absence, the communication was read by Dr. Juan A. del Regato, the subject being on the effects of the combined actions of x-rays and of a chemical carcinogen on the rat's liver. Dr. Gilbert H. Fletcher, Chair-

man for the afternoon session, called on Dr. Joseph L. Morton to act as moderator for a panel discussion on radiosensitivity and radiocurability as judged by microscopic techniques. Dr. Arthur T. Hertig spoke on the correlation of radiosensitivity and histopathology of cervical cancer. Dr. Ruth M. Graham presented the cytologic prognosis of cancer of the cervix and Dr. Saul B. Gusberg related his experience on radiosensitivity testing of cervix cancer in terms of tumor response. The afternoon session continued with papers on the incidence and treatment of radiation necrosis of the oral cavity and on distant metastases from oral and pharyngeal cancer.

The usual highlight of the Annual Meeting is the Janeway Lecture. Dr. Edith H. Quimby of New York reviewed the life of Dr. Henry Harrington Janeway and introduced Dr. Norman McCormick of Windsor, Ontario, who in turn introduced his fellow Canadian, The Janeway Lecturer, Dr. Clifford L. Ash of Toronto, whose subject was "Oral Cancer—A Twenty-five Year Study." He paid tribute to his former teacher, Dr. Gordon E. Richards and to his surgical colleague Dr. Harold Wookey. Stress was placed on the cooperation between the various specialties in the successful management of oral cancer. He described in some detail the facilities afforded by The Ontario Cancer Institute in association with the Princess Margaret Hospital and Divisions of Biological and Physics Research. During the years 1929 through 1958, 1,837 cases or oral cancer were registered. Statistics for five year results were obtained on 1,624 patients from 1929 through 1955 representing a 96 per cent follow-up. The crude five year survival rates by site and stage were given in detail for cancer of the tongue, buccal mucosa, gingiva, floor of the mouth and palate. The over-all five year results for all sites and stages was 31.5 per cent. The treatment policy is primarily radiotherapy, cervical metastases being managed by radical neck dissection.

Later in the evening of the first day, a reception was sponsored by the Radium Chemical Company of New York on the attractive Terrace Pool of the Broadmoor Hotel. An enjoyable social hour was spent by the members and guests of the Society.

The second day's scientific session under the Chairmanship of Dr. Charles G.Stetson was filled with a variety of presentations. Drs. Chau, Fletcher, Rutledge and Dodd of Houston discussed complications in the high dose total pelvic irradiation of female cancer and Drs. Stander and Rhamey of Indianapolis presented findings on the intravenous pyelograms in patients with carcinoma of the cervix. Dr. Theodore R. Miller of New York City reported on 36 patients who were treated by hemipelvectomy for far advanced cancer. One of the interesting topics of present day research was given by Dr. Robert Rugh of Columbia University on low-level x-irradiation in the very early mammalian embryo. He used exposures of 5 to 15 r on 0.5 day to 1.5 day rat embryos and showed striking central nervous system anomalies. Dr. Edith H. Quimby in collaboration with Dr. John M. Hanford of Columbia University reported on cancer arising many years after radiation therapy of benign lesions in the cervical region. Out of 247 traced patients from a group of 650, there had developed 8 carcinomas of the thyroid, 3 of the pharynx and larynx and 1 of the parotid gland.

The latter part of the morning session included reports on the ten year evaluation of the treatment of bone metastasis from carcinoma of the breast and prostate using radioactive phosphorus and testosterone by Drs. J. G. S. Maxfield and J. R. Maxfield, Jr., characteristics of a cesium head-and-neck unit by Dr. M. Lederman of the Royal Marsden Hospital, the measurement of "build-up" on a curved surface exposed to beams of cobalt 60 and cesium 137 by Drs. R. S. Bush and H. E. Johns, clinical and biological studies of actinomycin D and radiations by Dr. G. J. D'Angio and medical support of air defense operations under radioactive fallout by Captain W. B. Dye, USAF, MC.

During the afternoon of the second day,

a visit was made in buses to the Air Force Academy. A reception was sponsored by the Society at the International Center preceding the Annual Banquet. Many of the past Presidents of the Society were seated at a head table and were introduced by Dr. Jesshill Love. With appropriate ceremony Dr. Clifford Ash was presented the Janeway Medal for his outstanding achievements. Following dinner, a colorful and exciting dance pageant was staged by the Koshare Indians, a group of Boy Scouts who have been well trained in the folk-lore of many ancient tribal dances. This outstanding entertainment was followed by music and dancing for members and guests of the Society.

Dr. Victor A. Marcial was Chairman of the third day's scientific session. A presentation on the Plummer-Vinson syndrome and postcricoid carcinoma was given by Dr. Folke Jacobsson of the Radiumhemmet. A board of inquiry was moderated by Dr. Milton Friedman in which Dr. Fernando G. Bloedorn of Baltimore presented a three year survival of a patient with an extensive peritonsillar carcinoma with metastases to the homolateral lymph nodes, Dr. T. A. Watson of Saskatoon showed a ten year cure of a giant-cell tumor of the cervical spine and Dr. James A. Martin of Dallas demonstrated treatment techniques on an eight year survival of a recurrent low-grade fibrosarcoma of the vulva. Discussion of these cases was given by Drs. del Regato, Ennuyer, Lenz, and Lederman.

A group of papers relating to diagnostic and therapeutic considerations of brain tumors was next on the program. These communications included problems of instrumentation and technique of localization of brain tumors using radioiodinated human serum albumin, radiation therapy of brain tumors, irradiation of meningioma and glioblastoma multiforme. A three-dimensional radium reconstruction device was reported by Dr. Jerome M. Vaeth and a color motion picture on operating room techniques of radium implants was shown by Dr. Fernando G. Bloedorn. The General Electric Company sponsored the appearance of Dr. Bernard Pierquin from the Institut Gustave Roussy in Paris who presented material on laminagraphy in the dosimetric calculations of interstitial curie therapy.

One of the striking statistical reports of the meeting was given by Dr. Sigvard Kaae from the Radium Center in Copenhagen. Five year results on two random series of breast cancer treated by simple mastectomy and postoperative irradiation (McWhirter) versus extended radical mastectomy (Dahl-Iverson) indicated no significant difference. The last paper on the program by Drs. A. Ennuyer and J. P. Bataini on five and ten year results on combined surgery and radiotherapy in the treatment of cancer of the laryngeal vestibule was read by Dr. Juan A. del Regato.

The Forty-third Annual Meeting of the American Radium Society in the attractive setting of Colorado Springs was successful from the standpoints of quality of its scientific program, attendance, good weather and social events. Dr. Jesshill Love and his energetic committees can well be proud of their efforts.

Harry Hauser, M.D.



SAMUEL W. DONALDSON
1891-1961

DOCTOR SAMUEL W. DONALDSON, aged sixty-nine, Chief Radiologist at St. Joseph Mercy Hospital, Ann Arbor, Michigan, died of a heart attack in the early morning hours, Saturday, April 8, 1961.

Doctor Donaldson was born in Rockford, Tennessee, September 14, 1891. His early education was received in private and public schools in Asheville, North Carolina, and Knoxville, Tennessee, and he graduated from Knoxville High School in 1907. Following his graduation from high school, he entered the University of Alabama,

where he remained for one semester. He then entered the University of Tennessee and received his Bachelor of Arts degree in 1912. Entering the University of Michigan, he received his M.D. degree in 1916. Following this he interned at the German Hospital, which is now known as Lenox Hill Hospital, New York City, 1916–1917. He received his radiological training under the late Preston M. Hickey, at the University of Michigan Medical School, Ann Arbor, Michigan.

At the time of his death, Doctor Donaldson was associated with St. Joseph Mercy Hospital since 1923. In addition to his active services as Chief Radiologist in St. Joseph Mercy Hospital, he was also Radiologist at the Ypsilanti State Hospital, Ypsilanti, Michigan, having held that position since 1946. Doctor Donaldson organized the X-ray Department at St. Joseph Mercy Hospital, which he headed for thirty-eight years.

He became nationally known for his contributions in the field of radiology. Throughout his entire medical career as a radiologist, he participated actively in the organizations of radiology with which he was associated. His services and contributions to the advancement of radiology were outstanding. It has been said that "People acquire a varying mental equipment for the journey of life," and this may be said of Sam Donaldson.

He had a particular flair for statistics, and among his hobbies were medical economics and forensic medicine. His statistical knowledge concerning radiological departments, cost per patient, per film, and the personnel required in the radiological departments to handle a given number of patients per day was set forth in a paper which provoked an enormous amount of interest and comment following its publication. Indeed, it is accepted as a manual for the organization of radiological departments. He also published a text on medicolegal practice, which passed through its second edition.

His love for cooking led him to compile eleven cook books in one, called "Ann Arbor Cook Books, 1899–1959."

He maintained a great interest in sports, and his close friends maintained they were sure he could answer the $64,000 question concerning baseball in its various phases and its practice.

Doctor Donaldson pursued many activities, authoring numerous papers in his field, serving as a physician, participating in numerous professional societies, training x-ray technicians, and serving the community with a "side line." It involved x-raying animals as "a courtesy for neighbors who otherwise couldn't get this type of service." He used to say "My veterinary practice has made firm friends for medicine, for radiology, and for doctors."

Doctor Donaldson was the recipient of many honors, among which was a fellowship in the American College of Radiology, and he was awarded in 1955 its Gold Medal for distinguished service in the advancement of radiology.

He was a member of the American Medical Association, American Roentgen Ray Society and at the time of his death was Second Vice-President of this Society, a member of the Radiological Society of North America, the Detroit Roentgen Ray and Radium Society, and the Michigan State Medical Society. At the time of his death he also was Chairman of the Medicolegal Committee of the Radiological Society of North America, Director of the Professional Bureau and a board member of the American College of Radiology. He was a former editor of the Phi Rho Sigma journal. He was a member and Past-President of the Washtenaw County Medical Society, Past-President of the Detroit Roentgen Ray and Radium Society, and Past-President of the Michigan Association of Radiologists.

Doctor Donaldson was a major in the Medical Corps in World War I.

In 1919, he married Kathryn P. Overman of Danville, Illinois. She died in 1954, and he married Margaret L. Lowe of Seattle, Washington, in 1957, who sur-

vives him. He is also survived by a daughter, Mrs. G. B. (Mary E.) Hotchkiss, Washington, D. C., and a son, John G., Madison, Wisconsin, and six grandchildren.

Doctor Donaldson had a host of friends throughout this country and abroad, who will be grieved to learn of his untimely death.

Lawrence Reynolds, M.D.

Vol. 86, No. 2

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*
Donn G. Mosser, M.D., *Associate Director*

Titles and Abstracts of Courses Offered
Sixty-Second Annual Meeting
Deauville Hotel, Miami Beach, Florida
September 26 to September 29, 1961

The Instruction Courses for the Sixty-Second Annual Meeting of the American Roentgen Ray Society will all be of ninety minutes' duration and will be held between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M. on Friday. No other official activity has been scheduled for this time.

Seventeen rooms are available for the courses and one course will be offered in each room each afternoon of the Annual Meeting. A total of sixty-four instructors will give sixty-eight courses during the four days.

Full information will be available at the desk of the Section on Instruction, which will be located in the General Registration Area on Monday afternoon and from 8:30 A.M. until 4:30 P.M. Tuesday, Wednesday, Thursday and Friday.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only, except Course 305.

Following the list of titles and abstracts in this issue of the Journal there is a pink order sheet. First, second and third choices for each period should be indicated on the order sheet. It is possible for one to attend a total of only four periods of instruction. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized. It is estimated that the number who can attend each course each day will range between 30 and 100 persons depending on room size.

Reservations for the courses will be made in the order in which the pink order sheets are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $2.00 per period. Full-time graduate students in radiology will be admitted *without fee*, but they will be required to make application for the courses they wish to attend.

Prior to September 12, 1961, the order forms should be sent to the Director, Dr. Harold O. Peterson, 1995 West County Road "B", St. Paul 13, Minnesota. After September 12, 1961 the order forms should be sent to Dr. Harold O. Peterson, Deauville Hotel, Miami Beach, Florida.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 12, 1961. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the Registration Desk of the Section on Instruction, Monday afternoon, September 25, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An attendant will be at the door of each conference room to collect the tickets.

THE FACULTY

1. Kurt Amplatz, M.D., Assistant Professor of Radiology, University of Minnesota Medical School, Minneapolis, Minnesota.
2. Wilbur Bailey, M.D., Chairman of the Judiciary Committee, American College of Radiology, Los Angeles, California.
3. Robert P. Barden, M.D., Department of Radiology, Chestnut Hill Hospital; Associate Professor, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.
4. Earl Barth, M.D., Past President of the American College of Radiology, Professor and Head of the Department of Radiology, Northwestern University School of Medicine, Chicago, Illinois.
5. Frederic C. Bartter, Chief of the Endocrinology Branch of the National Heart Institute, Bethesda, Maryland.
6. Frank Batley, M.B., Ch.B., D.M.R.T., Director of Radiation Therapy, Syracuse Memorial Hospital, Syracuse, New York.
7. Fernando G. Bloedorn, M.D., Professor and Head of the Division of Radiotherapy, University of Maryland Hospital, Baltimore, Maryland.
8. Wallace D. Buchanan, M.D., Chairman of the Committee on Hospital Standards, American College of Radiology, South Bend, Indiana.
9. John A. Campbell, M.D., Chairman, Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.
10. G. J. D'Angio, M.D., X-ray Therapy Department, Children's Hospital Medical Center, Boston, Massachusetts.
11. Joseph N. Debbas, M.D., Department of Radiology, Hahnemann Medical College, Philadelphia, Pennsylvania.
12. Juan A. del Regato, M.D., Director, Penrose Cancer Hospital, Colorado Springs, Colorado.
13. Gerald D. Dodd, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
14. Charles T. Dotter, M.D., Professor and Chairman, Department of Radiology, University of Oregon Medical School, Portland, Oregon.
15. Robert L. Egan, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
16. Bernard S. Epstein, M.D., Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York.
17. Lewis E. Etter, M.D., Director, Department of Radiology, The Falk Clinic; Consultant, Western Psychiatric Institute and Clinic; Professor of Radiology, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.
18. William R. Eyler, M.D., Chairman, Department of Radiology, Henry Ford Hospital, Detroit, Michigan.
19. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.
20. Leo S. Figiel, M.D., Department of Radiology, The Grace Hospital, Detroit, Michigan.
21. Nathaniel Finby, M.D., Director of the Department of Radiology, St. Luke's Hospital, Morningside Heights, New York, New York.
22. Harry W. Fischer, M.D., Associate Professor of Radiology, Department of Radiology, University Hospitals, State University of Iowa, Iowa City, Iowa.
23. Robert H. Freiberger, M.D., The Hospital for Special Surgery, Margaret M. Caspary Clinic, New York, New York..
24. Brit B. Gay, Jr., M.D., Department of Radiology, Emory University, Atlanta, Georgia.
25. Bertram R. Girdany, M.D., Director, Department of Radiology, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania.
26. John Hale, Ph.D., Associate Professor of Radiological Physics, University of Pennsylvania, Philadelphia, Pennsylvania.
27. Theodore F. Hilbish, M.D., Chief, Diagnostic X-ray Department, The Clinical Center, Department of Health, Education and Welfare, Bethesda, Maryland.
28. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Professor of Clinical Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
29. Harold G. Jacobson, M.D., Head of the Division of Diagnostic Radiology of Montefiore Hospital; Professor of Clinical Radiology, New York University College of Medicine; Senior Consultant in Radiology to the Veterans Hospital in the Bronx, New York, New York.
30. John Kirkpatrick, M.D., Department of Radiology, St. Christopher's Hospital, Philadelphia, Pennsylvania.
31. E. C. Klatte, M.D., Associate Professor of Radiology, Chief of Pediatric Radiology, Indiana University Medical Center, Indianapolis, Indiana.
32. Simon Kramer, M.D., Co-Director, Radiation Therapy Department, and Associate Professor of Radiology, Jefferson Medical College, Philadelphia, Pennsylvania.
33. Richard G. Lester, M.D., Professor and Chairman, Department of Radiology, Medical College of Virginia, Richmond, Virginia.
34. Emanuel J. Levin, M.D., Director of Radiology, Maimonides Hospital of Brooklyn; Professor of Radiology, State University of New York, Brooklyn, New York.
35. Alexander Stewart Macmillan, Jr., M.D., Director, Department of Radiology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.
36. Victor A. Marcial, M.D., Director, Cancer Control, Department of Public Health; Director,

Department of Radiotherapy, Hospital Gonzalez-Martinez; Professor of Radiotherapy, University of Puerto Rico Medical School, San Juan, Puerto Rico.

37. Richard H. Marshak, M.D., Associate Radiologist, Mt. Sinai Hospital, New York, New York.
38. Charles L. Martin, M.D., Director, Martin X-ray and Radium Clinic; Clinical Professor of Radiology, Southwestern University Medical School, Dallas, Texas.
39. Gerald M. McDonnel, M.D., Department of Radiology, University of California Medical Center, Los Angeles, California.
40. Harry Z. Mellins, M.D., Professor and Chairman, Department of Radiology, State University of New York, Brooklyn, New York.
41. William T. Meszaros, M.D., Director, Department of Diagnostic Radiology, Cook County Hospital, Chicago, Illinois.
42. Russell H. Morgan, M.D., Radiologist in Chief, The Johns Hopkins Hospital, Baltimore, Maryland.
43. Walter T. Murphy, M.D., Director, Department of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo, New York.
44. Sidney W. Nelson, M.D., Professor and Chairman, Department of Radiology, The Ohio State University Medical School, Columbus, Ohio.
45. Charles M. Nice, Jr., M.D., Professor and Chairman, Department of Radiology, Tulane University School of Medicine, New Orleans, Louisiana.
46. James J. Nickson, M.D., Chairman, Department of Radiation Therapy, Memorial Hospital, New York, New York.
47. Robert S. Ormond, M.D., Department of Radiology, Henry Ford Hospital, Detroit, Michigan.
48. M. V. Peters, M.D., Ontario Cancer Institute, Toronto, Ontario, Canada.
49. Arthur J. Present, M.D., Chairman of the Committee on Public Relations, American College of Radiology, Tucson, Arizona.
50. Bernard Roswit, M.D., Chief, Radiation Therapy Service, Veterans Administration Hospital, Bronx, New York.
51. Philip Rubin, M.D., Chief, Division of Radiotherapy, Department of Radiology, University of Rochester School of Medicine, Rochester, New York.
52. Eugene L. Saenger, M.D., Associate Professor of Radiology, University of Cincinnati, Cincinnati, Ohio.
53. Jose Sala, M.D., Chief Radiotherapist, Ellis Fischel State Cancer Hospital, Columbia, Missouri.
54. Richard Schatzki, M.D., Chief, Department of Radiology, Mount Auburn Hospital, Cambridge, Massachusetts; Clinical Associate in Radiology, Harvard Medical School, Boston, Massachusetts.
55. William H. Shehadi, M.D., Director, Department of Radiology, United Hospital, Port Chester, New York.
56. Glenn E. Sheline, M.D., Assistant Radiotherapist and Associate Professor of Radiology, University of California Medical Center, San Francisco, California.
57. Edward B. Singleton, M.D., Director, Department of Radiology, St. Luke's and Texas Children's Hospitals, Houston, Texas.
58. Justin J. Stein, M.D., Chief, Radiation Therapy Division and Professor of Radiology, University of California Hospitals, Los Angeles, California.
59. William J. Tuddenham, M.D., Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
60. T. J. Wachowski, M.D., Chairman of the Board of Chancellors, American College of Radiology, Wheaton, Illinois.
61. H. Stephen Weens, M.D., Professor and Head, Department of Radiology, Emory University School of Medicine, Atlanta, Georgia.
62. Marvin M. D. Williams, Ph.D., Professor of Biophysics, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.
63. Martin H. Wittenborg, M.D., Children's Hospital; Assistant Clinical Professor, Harvard Medical School, Boston, Massachusetts.
64. George T. Wohl, M.D., Associate Professor of Radiology, Temple University School of Medicine; Chairman, Department of Radiology, Philadelphia General Hospital, Philadelphia, Pennsylvania.

DESCRIPTION OF COURSES

COURSE 101

Tuesday

NATHANIEL FINBY, M.D.
New York, New York

Male Cystourethrography

A general survey of male cystourethrography will stress normal and pathologic roentgenographic anatomy. Congenital and acquired diseases of the lower urinary tract will be demonstrated on cystourethrograms.

COURSE 102
and
COURSE 202

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-urinary Tract of Infants and Children

Course 102. Probably the most important examination in Pediatric Radiology with the highest yield of positive returns is intravenous urography, and yet, in spite of its importance, it is a study that is frequently poorly executed. There are a multitude of reasons for this. The speaker will attempt to show his own problems with this procedure and how, by working at it over a long period of time, he arrived at a technique which nine out of ten times results in a diagnostic study. This technique will include various methods of reducing the roentgen exposure to the gonads. It will also illustrate the importance of obtaining a voiding urethrogram in the male as one of the final steps in the procedure.

Having presented the technique of intravenous urography, the speaker will then attempt to illustrate his own evolutionary understanding of posterior-urethral valves, ectopic ureteroceles, absence of the abdominal musculature, and of as many other congenital anomalies as time will permit.

Mediastinal Mass Lesions in Infants and Children

Course 202. Mass lesions occurring in the mediastinum can be diagnosed with a high degree of accuracy simply by knowing whether they occur in the front, in the middle or in the back. This course will attempt to give a comprehensive discussion of most of the mass lesions which occur in the mediastinum. There are a few differences between the younger age group and the older age group and these differences will be discussed.

COURSE 103

Tuesday

RUSSELL H. MORGAN, M.D.
Baltimore, Maryland

Screen Intensification and Cineradiography

This course will deal with the physics and technical problems associated with screen intensification and cineradiography. The discussion will consider those aspects of x-ray image formation and perception which are important in the fields of screen intensified fluoroscopy and motion picture recording. Particular attention will be given to the practical problems associated with the use of various intensifier and cineradiographic systems in clinical radiology.

COURSE 104

Tuesday

PHILIP RUBIN, M.D.
Rochester, New York

The Development of a Dynamic Classification of Bone Dysplasias

There is a compelling logical order to the way normal bones model. From a study of the development of normal bone growth and an analysis of the physiologic processes, one may be able to provide the key to unlock the abnormal pathophysiology in dysplastic bone diseases. An attempt is made to set up criteria for true bone dysplasias, based on an understanding of experimental and clinical pathophysiology. It is hoped that one can develop, on this basis, a dynamic classification for this seemingly complicated group of diseases. The differential diagnostic points of different bone dysplasias will be emphasized.

COURSE 105

Tuesday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

This course consists of an analysis of the sources of error in roentgen diagnosis, a critique of existing concepts of "film quality," and proposals for modified radiographic and interpretative techniques designed to increase the accuracy of the method.

The inadequacy of "contrast" and "unsharpness" as criteria for evaluating the diagnostic usefulness of roentgenograms will be discussed and new criteria will be presented.

The significance of physical factors said to limit the "quality" of roentgenograms will be considered and the indications for so-called "high-kilovoltage" and "supervoltage" radiography as well as for the

use of the "micro-focus" roentgen-ray tube will be specifically noted.

Psychophysiologic factors which limit the accuracy of roentgen diagnosis will be emphasized, and recent experimental studies relating to problems of visual search and image organization will be presented. Also, control of view-box and "surround" illumination, control of glare, and the selection of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 106
and
COURSE 206

Tuesday and Wednesday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses

Course 106. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and various skull types. Analysis of individual bone components in producing the various lines and shadows seen in plain roentgenograms of the skull. This analysis will be applied to a study of the sphenoid, frontal, ethmoid, parietal and occipital bones in various projections.

Course 206. Detailed roentgen anatomic analysis of the paranasal sinuses and mastoids with description of roentgenographic techniques employed. Study of the roentgen features of the middle cranial fossa and their relation to the sphenoid sinuses. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. In conclusion, the maxillofacial components of the cranium will be examined, including the lacrimal canals and soft tissue delineation.

COURSE 107

Tuesday

H. STEPHEN WEENS, M.D.
Atlanta, Georgia
BRIT B. GAY, JR., M.D.
Atlanta, Georgia

Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes

I. Methods of roentgenologic study of pulmonary circulation: (a) roentgenoscopy with image amplification, (b) routine roentgenography, (c) laminagraphy, (d) pulmonary angiography and (e) cineroentgenography.

II. Basic function of pulmonary circulation: normal pulmonary arterial and venous pressure; normal pulmonary blood flow.

III. Increased pulmonary blood flow. Definition. Etiology: left to right shunts; admixture lesions. Roentgenologic signs. Pulsations of the pulmonary arteries in increased pulmonary blood flow.

IV. Decreased pulmonary blood flow. Definition. Etiology: left to right shunts with pulmonary stenosis or atresia. Roentgenologic signs. Roentgenologic appearance of collateral circulation to the lungs through bronchial arteries or anomalous arteries. Pulsations of pulmonary arteries in decreased pulmonary blood flow. Special consideration of pulmonary vasculature changes and pulsations in isolated pulmonary valvular stenosis.

V. Pulmonary venous hypertension—pulmonary edema. Roentgenologic signs. Significance of Kerley's (septal) lines. Special consideration of pulmonary venous changes in mitral stenosis. Importance of minimal signs of pulmonary interstitial edema as an indication of acute myocardial infarction.

VI. Pulmonary arterial hypertension. Definition. Etiology: roentgenologic signs. Pulsations of pulmonary arteries in pulmonary hypertension.

The roentgenologic changes of the pulmonary circulation will be illustrated by cinefluorograms.

COURSE 108
and
COURSE 208

Tuesday and Wednesday

EMANUEL J. LEVIN, M.D.
Brooklyn, New York

The Fundamentals of Bone Disease

There are characteristic radiologic patterns produced by diseases of the bones which permit a definite and accurate diagnosis. The reactions in the soft tissues, periosteum and cartilage are often of greater differential value than the changes in bone. Furthermore, many osseous diseases have peculiarities of destruction, production and localization which in themselves are of diagnostic significance. The de-

tection and correct assessment of the varied roentgen patterns will permit an accurate diagnosis in a high percentage of cases.

An analytic approach to the differential diagnosis of diseases of the bones will be presented with emphasis placed on fundamental aspects.

I. Explanation and description of terms
   A. Soft tissue reactions
   B. Periosteal reactions
   C. Osseous reactions
II. Tumors
   A. Benign
   B. Malignant
III. Infections
   A. Acute
   B. Chronic

COURSE 109
and
COURSE 209

Tuesday and Wednesday

ALEXANDER STEWART MACMILLAN, JR. M.D.
Boston, Massachusetts

Sinuses; Mastoids, Lateral Neck and Larynx

Course 109. Sinuses.
Course 209. Mastoids, lateral neck and larynx.

COURSE 110
and
COURSE 210

Tuesday and Wednesday

SIDNEY W. NELSON, M.D.
Columbus, Ohio

Bronchography

Course 110. The radiologist who can properly perform and interpret bronchograms is in a position where he can make outstanding contributions to the diagnosis and understanding of many chest diseases. The proper therapy for any disease which affects the bronchial tree is often dependent upon the accurate determination of the nature and extent of the bronchial involvement. Inasmuch as the interpretation of bronchograms requires considerable knowledge of the normal anatomy of the tracheobronchial tree and some of the common anatomic variations, the pertinent anatomy will be discussed and illustrated. A good knowledge of bronchial anatomy will enable the radiologist to systematically identify each of the major segmental bronchi, because of his knowledge of the expected distribution of each of these bronchi. The identification of each of the major segmental bronchi is extremely important in interpretation.

Once the basic bronchographic anatomy has been considered, the details of the bronchographic technique will be described. This will include the method of performing topical anesthesia, introduction of intranasal catheter, method of injecting contrast medium in order to assure systematic filling of the desired segmental bronchi, proper positioning of patient for filling of each segment, and the spot filming techniques employed to thoroughly record the filling of the desired segmental bronchi. This portion of the discussion will also include a description of some of the characteristic features of various contrast materials. The advantages of fluoroscopically controlled spot filming in comparison with conventional large film techniques will be emphasized. Good bronchographic technique can be mastered with little effort and can be quickly performed by the radiologist and his technical staff, thus precluding the necessity for resorting to the "team" approach which is often so time-consuming and frustrating in large busy hospitals.

Course 210. During the second session, the majority of the different types of bronchopulmonary pathology will be discussed including the following: 1. Chronic bronchitis: a. spasm; b. excessive secretions; c. dilated mucosal glands; d. destruction of mucosa. 2. Emphysema. 3. Segmental bronchiectasis. 4. Cystic lesions. 5. Bronchogenic carcinoma. 6. Pulmonary "coin" lesions. 7. Bronchial adenoma. 8. Erosion of bronchi by adjacent calcified lymph nodes. 9. Inflammatory strictures. 10. Bronchial relocation following atelectasis of surgical removal.

The value of bronchography in the differential diagnosis of many bronchopulmonary diseases will be emphasized throughout the course, as for instance, atelectasis due to bronchogenic carcinoma or other intrabronchial lesions. Segmental bronchiectasis with associated chronic pneumonic changes may mimic neoplasms on plain chest roentgenograms, but can be clearly identified by bronchographic methods. Chronic granulomas and other infections can frequently be identified by virtue of the bronchiectatic changes produced in adjacent bronchi, thus helping to differentiate these diseases from neoplastic processes. Certain pulmonary diseases have very specific bronchographic appearances; *e.g.*, bronchial adenomata, polycystic lung, etc.

It is hoped that this presentation will encourage radiologists to perform this extremely useful roentgenographic procedure more frequently in the future. In so doing, they will be able to make more significant contributions to the diagnosis and treatment of the patients referred to them for study by their colleagues who are not radiologists.

COURSE 111

Tuesday

MARTIN H. WITTENBORG, M.D.
Boston, Massachusetts

Pelvis and Hips in Infancy and Early Childhood

Fundamentals of the roentgen interpretation of the growing pelvis and hips will be presented and illustrated as they apply in the diseases commonly encountered in this age group.

COURSE 112

Tuesday

ROBERT L. EGAN, M.D.
Houston, Texas

Over Five Years' Experience with Mammography

Over five years' experience has been gained with soft tissue roentgenography of the breast in a tumor institute with an active breast service. These studies have proved a highly accurate and valuable adjunct to the clinical and pathologic evaluation of breast lesions.

A simple and readily adaptable roentgenographic technique will be presented. Cases illustrative of typical benign and malignant disease will be shown and the diagnostic features of each described and tabulated. Differential diagnosis, sources of error, limitations of the procedure and the clinical and pathologic implications of the method will be discussed.

The results of a statistical analysis of the first 1,000 consecutive examinations (all with a follow-up from one to five years) will be reviewed. The error in 245 cases of malignant disease was 0.82 per cent.

COURSE 113
and
COURSE 310

Tuesday and Thursday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

Course 113. 1. Technique, including a discussion of the various barium preparations. 2. Regional enteritis. 3. The malabsorption syndrome, including sprue, lymphosarcoma and Whipple's disease. 4. Problems in the interpretation of functional changes. 5. Inflammatory lesions of the duodenum, excluding duodenal ulcers.

The Small Intestine and Colon

Course 310. A-1. Infarction of the small bowel. 2. Tumors. 3. Collagen diseases.

B-1. Inflammatory lesions of the colon.

COURSE 121

Tuesday

JUAN A. DEL REGATO, M.D.
Colorado Springs, Colorado

El Papel de la Radioterapia en el Tratamiento del Cancer

(This Course Will Be Given in Spanish)

La cirujía y la radioterapia tienen indicaciones precisas en el tratamiento del cáncer en las que una ú otra son el tratamiento de elección.

Además, existen ciertas posibilidades de fructrosa asociación de ambas disciplinas en beneficio del enfermo.

La radioterapia, como tratamento paliativo de enfermos incurables es, de por sí, un método indispensable en el tratamiento de estos enfermos.

COURSE 122

Tuesday

EUGENE L. SAENGER, M.D.
Cincinnati, Ohio

Diagnostic Uses of Radioisotopes (Other Than for Thyroid Disease)

This course will discuss the clinical uses of radioisotopes utilizing a scintillation probe, a scintillation well counter, a scanner and single channel pulse height analyzer.

The topics to be included are as follows:

I. Brief remarks on standardization of the instrument
II. Hematologic tests
  A. Iodinated albumin
  B. Chromium 51
    1. Red cell mass
    2. Red cell life span
    3. Sequestration
  C. Iron 59
    1. Iron turnover
    2. Iron utilization
  D. Simultaneous techniques utilizing Cr^{51} and Fe^{59}
III. Vitamin B_{12} (Co^{60})—Pernicious anemia
IV. I^{131} labeled fats and fatty acids
V. P^{32} in detection of eye tumors
VI. The scanogram
  A. Liver
  B. Chest
VII. I^{131} labeled PVP for evaluation of intestinal protein loss

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COURSE 123

Tuesday

MARVIN M. D. WILLIAMS, Ph.D.
Rochester, Minnesota

The r, rad and rem in Specification of Dose

Three different units are used in expressing dosage in radiation therapy. These are the exposure dose, which is measured in r, the absorbed dose, which is measured in rads, and the biologic dose, which is measured in rems. The meaning of these three units, their use, and methods of measuring and calculating them will be discussed.

At the present time, sufficient data are not available to calculate dosage in rems. To calculate the dosage in rads involves the use of conversion factors that often are uncertain. The determination of the exposure dose, which is the basis for calculating the absorbed dose, may have a rather high probable error. The combination of these uncertainties and probable errors often makes a statement of dose in rads of questionable value.

COURSE 124

Tuesday

G. J. D'ANGIO, M.D.
Boston, Massachusetts

Radiation Therapy in the Management of Children with Tumors

The discussion will include a description of the most common pediatric-age neoplasms with emphasis on their peculiarities and the unpredictable clinical course followed by some of these tumors. Treatment as practiced at the Children's Hospital Medical Center will be reviewed as will the results of therapy and the late effects of irradiation delivered to infants and children.

COURSE 201

Wednesday

HARRY W. FISCHER, M.D.
Iowa City, Iowa
GERALD D. DODD, M.D.
Houston, Texas
JOSEPH N. DEBBAS, M.D.
Philadelphia, Pennsylvania

Lymphography

The historic development of lymphography will be briefly described. The various techniques which have been utilized for lymphography will be discussed and recommendations made for the best available technique at the present time. Since visualization of the lymphatic structures depends upon specialized characteristics in contrast material, the presently available materials will be commented upon and their advantages and deficiencies compared. Suggestions toward the development of a better contrast material will be made. The anatomy of the normal lymphatic structures will be then described as a base for the discussions of pathologic findings which will follow.

The use of lymphography in diagnosing and evaluating the extent of neoplastic disease in the lymph nodes will be discussed by Dr. Dodd based on his clinical experiences with the method at the M. D. Anderson Hospital and Tumor Institute. The use of lymphography and evaluation of the edematous leg will be discussed by Dr. Debbas.

A summary of the clinical experience will then be made with additional comments on the possible therapeutic applications of the same technique used in the diagnostic field. Time will be allowed for discussion between the three major participants and for questions from the floor.

COURSE 203

Wednesday

CHARLES M. NICE, JR., M.D.
New Orleans, Louisiana

Roentgenographic Manifestations of Collagen Diseases

Many conditions have been included under the term of collagen diseases. In general, diseases involving wide-spread changes in connective tissue or in small vessels have been called collagen diseases at one time or another.

Most writers include systemic lupus erythematosus, polyarteritis nodosa, systemic scleroderma, dermatomyositis, and rheumatic fever in the collagen disease group. Rheumatoid arthritis is considered by many to be a phase of the development of the major collagen diseases described above.

The roentgenographic findings in the above conditions will be described, the material being basically related to a study of 109 patients at the University of Minnesota Hospitals, later followed by an extension of this study at Charity Hospital in New Orleans. Combinations and sequences of findings that will help one to suspect collagen diseases will be emphasized in the presentation.

COURSE 204
and
COURSE 304

Wednesday and Thursday

HAROLD G. JACOBSON, M.D.
New York, New York

Malignant Bone Tumors—Primary and Secondary

Course 204. In this presentation the roentgen characteristics of the important malignant bone tumors—both primary and secondary—will be illustrated. These will include: (1) osteogenic sarcoma, (2) Ewing's sarcoma, (3) chondrosarcoma, (4) sarcoma with Paget's disease, (5) fibrosarcoma, (6) chordoma, (7) synovioma, (8) myelomatosis, (9) reticulum cell sarcoma, (10) Hodgkin's disease, (11) lymphosarcoma, (12) leukemia, (13) metastatic carcinoma.

In all instances the roentgen findings will be evaluated against the background of a discussion of the underlying pathologic changes for an adequate understanding of these roentgen abnormalities.

The Vaterian Segment: Normal and Abnormal

Course 304. The roentgen anatomy of the normal vaterian structures *in vitro* will be described utilizing cadaver material. The *in vivo* appearance of the normal vaterian papillae in the upper gastrointestinal tract examination will be illustrated and emphasized after a brief description of the various methods of the roentgen examination of the vaterian region. There will then follow the discussion of some of the important disease entities involving the vaterian area. The section on abnormalities will be dealt with against the background of a classification of enlargement of the papilla of Vater divided into two major categories: centric and eccentric enlargements. The centric enlargements will include those associated with common duct stone, acute and chronic relapsing pancreatitis, and carcinoma of the papilla and ampulla of Vater. The eccentric enlargements will be demonstrated mainly by cases with peptic ulcer disease in the proximal duodenum. The differential diagnosis of the various disease states causing these enlargements will be emphasized and illustrated with the aid of an outline long in use for evaluating abnormalities of the vaterian segment. The roentgen findings in carcinoma of the head of the pancreas will also be briefly described, particularly as these changes relate to the vaterian segment.

COURSE 205
and
COURSE 405

Wednesday and Friday

CHARLES T. DOTTER, M.D.
Portland, Oregon

The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department

In association with contrast cardiovascular visualization studies, myocardial anoxia may lead to cardiac arrest or ventricular fibrillation. Unless serious coronary occlusion, aortic stenosis or insufficiency is present, there is an excellent chance of effective resuscitation.

Cardiac arrest. This is most commonly encountered in association with general anesthesia and carries a better prognosis than does ventricular fibrillation. The treatment is prompt adoption of measure promoting an increased flow to the myocardium of oxygenated blood. Mouth-to-mouth resuscitation and anteroposterior compression of the lower sternum against the spine provide effective means for accomplishing this purpose. If a normal heartbeat does not return, the use of a pacemaker may be considered. Thoracotomy is not only unnecessary, it is far less likely to result in survival of the patient.

Ventricular fibrillation. Ventricular fibrillation is prone to occur in people with diminished coronary flow. When it occurs as a product of uncorrected myocardial anoxia, defibrillation will not be successful. Often ventricular fibrillation following coronary occlusion is due to excitation of a small infarct rather than diffuse myocardial anoxia. Here, prompt resuscitation is very likely to be lifesaving. Resuscitation consists of properly applied compression of the lower sternum against the vertebral column. This form of resuscitation is simple to perform and—as will be demonstrated by pressure records comparing both closed and open techniques in the same patient—more effective than direct open-chest cardiac massage. Details will be cited covering several emergency resuscitations including four open-chest cardiac massages with two survivals and one protracted resuscitation wherein left ventricular function was provided by several members of the Radiology Department during a two and one-half hour period. A simple, low-cost (homemade) defibrillator was repeatedly employed with success.

Credit for the technique for closed-chest cardiac resuscitation properly goes to Drs. Kouwenhoven, Jude and Knickerbocker of Johns Hopkins University. This technique will become standard and should, therefore, be familiar to all physicians.

COURSE 207
and
COURSE 307

Wednesday and Thursday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy, physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 207: (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 307. (1) Anatomic variations in the pulmonary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 211

Wednesday

RICHARD SCHATZKI, M.D.
Boston, Massachusetts

Differential Diagnosis of Lesions at the Lower End of the Esophagus

The technique of the examination of the lower end of the esophagus will be described. Some of the problems in this area will be discussed, particularly those concerning esophagitis, cancer, lower esophageal ring and hiatus hernia.

COURSE 212

Wednesday

WILLIAM R. EYLER, M.D.
Detroit, Michigan
ROBERT S. ORMOND, M.D.
Detroit, Michigan

The Acute Abdomen

This course is designed primarily to review the roentgenologic manifestations of acute abdominal disease, particularly, as manifested on plain abdominal films. When appropriate for illustrative purposes, associated examinations with contrast materials will be presented. The following outline includes the subjects to be discussed:

1. Anatomy of the peritoneal cavity as shown by pneumoperitoneum.
2. Differentiation of the hollow viscera.
3. Vascular diseases: (a) aneurysms, dissecting and arteriosclerotic; (b) occlusive vascular disease; (c) blood dyscrasias; (d) polyserositis; (e) collagen diseases.
4. Inflammatory diseases of hollow viscera: (a) acute appendicitis, acute cholecystitis, gastritis, enteritis and cholecystitis; (b) regional enteritis; (c) tuberculosis; (d) generalized peritonitis; (e) localized abscesses.
5. Inflammatory diseases of solid viscera: (a) acute pancreatitis; (b) acute perinephric and obstructive urinary tract diseases.
6. Mechanical obstructions of the digestive tube: (a) neoplastic diseases; (b) extrinsic pressure; (c) special mechanical obstructions including intussusception, volvulus, hernia and band.
7. Traumatic conditions: (a) solid viscera; (b) hollow viscera.
8. Miscellaneous conditions causing or simulating the acute abdomen including sprue, neurologic disorders, toxic agents and endocrine malfunctions.

COURSE 221

Wednesday

GLENN E. SHELINE, M.D.
San Francisco, California

The Use of Radioisotopes in the Diagnosis and Treatment of Thyroid Disease

This course will deal with the uses of radioiodine in the diagnosis of thyroid dysfunction, in the treatment of thyrotoxicosis, and in the treatment of thyroid cancer. In the material on diagnosis, emphasis will be placed on the thyroid uptake curve with or without thyroid suppression or stimulation tests. The uses and limitations of thyroid scanning will be considered. Included in the treatment of thyrotoxicosis will be information regarding the occurrence of leukemia, thyroid adenomas and thyroid carcinomas subsequent to radioiodine therapy.

COURSE 222

Wednesday

JUSTIN J. STEIN, M.D.
Los Angeles, California

The Use of Megavoltage Therapy in the Treatment of Internal Cancer

This course will consider indications and contraindications for megavoltage therapy in the treatment of malignant disease. Supplemental therapy will be discussed. Treatment programs and end results at the University of California at Los Angeles will be presented.

COURSE 223

Wednesday

SIMON KRAMER, M.D.
Philadelphia, Pennsylvania

Radiotherapeutic Retreatment: To Do or Not To Do?

The radiologist is commonly confronted with the problems of retreating recurrent malignancies previously irradiated or of treating new lesions in areas previously irradiated.

The discussion will focus on principles to be observed in deciding on appropriate procedures and also on methods to be utilized. Specific problems will be discussed to elucidate the principles involved.

COURSE 224

Wednesday

CHARLES L. MARTIN, M.D.
Dallas, Texas

The Treatment of Pulmonary Metastases with Roentgen-Ray Therapy

Most radiologists take a pessimistic attitude toward the treatment of metastatic pulmonary lesions with irradiation because it is a well known fact that the administration of cancerocidal doses to both lungs can produce a fibrosis with a fatal outcome in a relatively short time. No doubt many are also unfamiliar with an excellent paper published by Evans and Leucutia in 1924 in which it was demonstrated that relatively small doses of roentgen rays caused many pulmonary metastases to regress without the production of lung damage.

The technique, which must be carefully carried out to be effective, requires only conventional equipment and a series of slides will be shown to illustrate results and technical pitfalls. This method is not curative but may give the acutely ill patient one to three years of comfortable living.

COURSE 225

Wednesday

VICTOR A. MARCIAL, M.D.
San Juan, Puerto Rico

Cancer of the Tongue: Its Therapeutic Management

Consideration will be given to the natural history and incidence of cancer of the tongue. Aspects of the problem to be discussed are: (1) factors determining method of treatment (radiotherapy or surgery); (2) factors in choice of type of radiotherapy (interstitial, interstitial plus external, isotopes used in place of radium); (3) different techniques of implantation; (4) dosimetry problems; (5) care of patients during and after implantation; (6) complications and their management; and (7) management of lymph node involvement.

COURSE 301

Thursday

ROBERT P. BARDEN, M.D.
Philadelphia, Pennsylvania

Interpretation of Radiologic Signs of Abnormal Pulmonary Function

Knowledge of some aspects of abnormal pulmonary physiology helps the clinical radiologist in two ways: he may diagnose many chest diseases more completely, and he may be able to evaluate the effect of disturbed pulmonary function on other systemic diseases.

This course will show examples of roentgenologic examinations which provide help in evaluating disturbances in ventilation, diffusion and arterial oxygenation.

COURSE 302

Thursday

THEODORE F. HILBISH, M.D.
Bethesda, Maryland
FREDERIC C. BARTTER, M.D.
Bethesda, Maryland

Roentgen Findings in Diseases with Abnormal Calcium Deposition

This course will present the roentgen manifestations of diseases associated with abnormal calcium deposition including such conditions as calcinosis universalis, myositis ossificans, hypoparathyroidism, collagen vascular diseases, etc.

The purpose will be to record the interesting and, at times, unusual roentgen findings associated with abnormal calcifications in the tissues. No attempt will be made to cover every entity causing abnormal calcification but the more interesting and informative conditions will be included. A brief discussion of the differential diagnosis will be given in each instance with the intent of affording the radiologist a quick source of identification as to etiology.

COURSE 303

Thursday

GERALD M. MCDONNEL, M.D.
Los Angeles, California

The Radiation Hazard

The trend in evaluation of the radiation hazard has been to the extreme of conservatism. Often the evaluation is performed by nonmedical persons and, if done within the medical profession, often not by the radiologist. Factual data relating natural radia-

tion hazards of the environment to man-made contamination of the biosphere (fall-out) will be given. Comparison of these values to the radiation dosage obtained in diagnostic radiographic procedures will be made. The implication of these data to the population at large and the responsibility of the radiologist in evaluating the information will be discussed.

COURSE 305

Thursday

EARL BARTH, M.D. (MODERATOR)
Chicago, Illinois
WILBUR BAILEY, M.D.
Los Angeles, California
WALLACE D. BUCHANAN, M.D.
South Bend, Indiana
ARTHUR J. PRESENT, M.D.
Tucson, Arizona
T. J. WACHOWSKI, M.D.
Wheaton, Illinois

Current Opinions from the American College of Radiology on Principles of Ethics and Economics

This course will cover the principles of medical ethics as they apply to radiologic practice in hospitals, offices and clinics. Many facts and figures compiled by the American College of Radiology will be presented.

It will be the purpose of this course to point out how the College can be most useful to its members and thus ultimately assist in developing the best possible radiologic practices. The course should be of special interest to the younger men.

Course 305 will start at 1:30 P.M. and will be held in Napoleon Room No. 3. No tickets will be required for this course.

COURSE 306
and
COURSE 406

Thursday and Friday

WILLIAM H. SHEHADI, M.D.
Port Chester, New York

Radiologic Study of the Biliary Tract

Although some pathologic conditions involve primarily the liver and others the extra hepatic biliary tract, radiologically as well as clinically, the biliary tract should be considered one unit. The anatomy, physiology and pathology of the biliary tract will be briefly reviewed, with special emphasis on a study of liver function tests and jaundice, bringing out the role they play in biliary tract visualization.

The course will encompass the technique of liver examination, liver function tests and jaundice, and a study of liver diseases which are radiologically significant.

Also the techniques of gallbladder and bile duct visualization, the relative value of the oral and intravenous methods, and an evaluation of contrast media used in biliary tract visualization will be covered. The commonly as well as the infrequently encountered diseases of the gallbladder and ducts and the so-called postcholecystectomy syndrome will be discussed from a radiologic point of view.

COURSE 308
and
COURSE 408

Thursday and Friday

BERNARD S. EPSTEIN, M.D.
New Hyde Park, New York

Myelographic Changes Associated with Spinal Osteophytosis

Course 308. The instructor's technique for myelographic examination and the normal radiographic anatomy of the spinal canal will be presented briefly. Variations in the architecture of the vertebrae affecting the available space in the spinal canal will be considered. The clinical and radiologic aspects of spondylosis deformans and concomitant degenerative changes in the intervertebral disks will be reviewed. The consequences of intraluminal protrusion of spurs and ridges into the spinal canal and the intervertebral foramina will be discussed.

The Radiographic Investigation of the Basilar Cisterns

Course 408. The normal anatomy of the basilar cisterns and their pneumoencephalographic demonstration will be reviewed, including mention of the diagnostic aid afforded by laminagraphy and positive contrast examinations. The changes produced by neoplastic lesions impinging upon the basilar cisterns will be discussed, correlating the cisternal abnormalities with those of the adjacent brain and cranium. The effects of various inflammatory changes also will be considered.

COURSE 309
and
COURSE 409

Thursday and Friday

RICHARD G. LESTER, M.D.
Richmond, Virginia

Angiocardiography in the Diagnosis of Operable Heart Disease

The place of angiocardiography and aortography in the diagnosis of many congenital cardiac lesions

and certain acquired diseases is now well established. In these sessions emphasis will be placed on those conditions that are amenable to surgical correction or amelioration. Correlation with postmortem anatomy will be provided in large part by comparison of angiocardiograms and specimens of individual patients.

Introduction to Angiocardiography

Course 309. This session will begin with a discussion of indications for angiocardiography. Methods and techniques will be presented. The equipment necessary for both forward and selective examinations will be noted and the relative merits of rapid-film units and cineradiographic apparatus discussed.

Methods for the analysis of angiocardiographic examinations will follow. Criteria for the diagnosis of the more common lesions will be given. Data of importance to the surgeon will be emphasized.

Advanced Angiocardiography

Course 409. A more detailed analysis of certain of the findings in lesions discussed in the previous session will be provided. In addition, there will be consideration of other lesions of relative rarity but of surgical significance. The place of angiocardiography as a research method will be touched upon.

Participants are invited to show and discuss problem cases during the second session.

COURSE 311
and
COURSE 411

Thursday and Friday

GEORGE T. WOHL, M.D.
Philadelphia, Pennsylvania
JOHN KIRKPATRICK, M.D.
Philadelphia, Pennsylvania

The Role of the Radiologist in Bone Tumor Diagnosis

There have been many recent advances in our knowledge of bone lesions. These make possible a more accurate roentgen diagnosis of many benign and malignant bone tumors. In this course a practical method of analyzing bone lesions useful to the radiologist will be presented.

A simplified pathologic correlation will be discussed. Many cases will be presented drawn from the files of the Philadelphia Orthopedic-Radiologic-Pathologic Conference. In the first session, the classification of bone tumors will be presented. The method of analyzing bone lesions will be reviewed. The various benign bone tumors will be presented for discussion. The second session will cover malignant bone tumors and lesions simulating bone tumors.

COURSE 312
and
COURSE 412

Thursday and Friday

JOHN A. CAMPBELL, M.D.
Indianapolis Indiana
E. C. KLATTE, M.D.
Indianapolis, Indiana

Cineradiography

That cineradiography is a useful research method and teaching aid has been established. It has also received wide acceptance as a practical clinical method in the cardiovascular, genitourinary, and gastrointestinal fields and is gaining popularity in orthopedic, neurologic, and certain other applications. Some type of image intensification must be utilized to permit an acceptable radiation dose to the patient. This may result in a reduction in desirable image detail and limitation in field size.

The diagnostic yields obtained with currently available equipment may be enhanced by a proper understanding of the inherent limitations of the method, the controllable technical factors, and the requirements for successful clinical adaptations. This course will present the technical and clinical considerations in cineradiography as outlined below.

Course 312. *Part I—Technical Considerations:* 1. Historical. 2. Current types of cine systems. 3. Roentgen-ray tube requirements. 4. Table adaptations. 5. Optical systems. 6. Cameras. 7. Image resolution, brightness and contrast requirements. 8. Exposure factors. 9. Kinescopic versus direct recording. 10. Film characteristics. 11. Processing. 12. Film projection and viewing. 13. Filing. 14. Radiation dose. 15. Control of image quality.

Course 412. *Part II—Clinical Considerations:* 1. Advantages and disadvantages of clinical cineradiography. 2. Clinical methodology of (a) cinecardioangiography, (b) cystourethrography, (c) orthopedic applications, (d) neuroroentgenology, (e) gastrointestinal applications, (f) gynecologic applications, (g) respiratory applications. 3. Future developments.

COURSE 313

Thursday

HARRY Z. MELLINS, M.D.
Brooklyn, New York

Nonobstructive Acute Abdominal Disorders

An analytic approach to the roentgenographic diagnosis of the nonobstructive acute abdominal disorders will be presented. Emphasis will be placed upon the plain film findings. The importance of serial or follow-up examinations will be stressed. The course will include inflammatory conditions, trauma, toxic conditions and vascular disorders.

COURSE 321

Thursday

JOHN HALE, Ph.D.
Philadelphia, Pennsylvania

A Diagnostic Physics Laboratory Course for Radiology Residents

Teaching of radiologic physics has tended to overemphasize the physics of therapy and radioactive isotopes. In an attempt to balance this situation, a one week long laboratory course in the physics of diagnostic radiology has been designed for residents at the Hospital of the University of Pennsylvania. This course will be described in some detail. The theoretic background and practical importance of each experiment will be discussed, and typical experimental results will be shown.

The course consists of the following experiments:

Radiography: (1) Basic measurements of the physical characteristics of a radiographic unit. (2) Film response characteristics. (3) Effect of radiographic equipment variables. (4) Effect of darkroom variables. (5) Unsharpness. (6) Perception of the visual image.

Fluoroscopy: (1) Basic physical measurements in fluoroscopy. (2) Contrast and detailed perception of: (a) a conventional fluoroscope, (b) an image intensifier.

COURSE 322

Thursday

M. V. PETERS, M.D.
Toronto, Ontario, Canada

Indications for Radiotherapy during the Course of Hodgkin's Disease

In this presentation the treatment of the new case of Hodgkin's disease according to stage will be reviewed and a suggested plan of management presented based on the findings on admission.

The treatment of recurrent disease is often more puzzling than the management of the newly diagnosed case. For this reason radiotherapy and chemotherapy will be discussed with regard to the more important sites of recurrence.

COURSE 323

Thursday

WALTER T. MURPHY, M.D.
Buffalo, New York

Carcinoma of the Vagina and Female Urethra

Primary carcinomas of the vagina and female urethra occur relatively infrequently. However, they should be of great importance to the radiation therapist because of their impressive radiocurability with resultant preservation of normal pelvic functions. Discussion will cover the clinical-pathologic aspects, the various radiotherapeutic techniques, radiation tolerance of the vulvo-perineal and contiguous pelvic tissues, and end results.

COURSE 324

Thursday

BERNARD ROSWIT, M.D.
Bronx, New York

Cancer Chemotherapy in Radiologic Practice

It is essential that radiologists keep well informed of newer developments in cancer chemotherapy in order to improve their management of patients with advanced disease. This course has, therefore, been planned by a radiologist for radiologists to discuss all available drugs, dosage, indications, clinical benefits, toxicity, complications and, above all, integration of drugs with the radiation treatments. A chemotherapy manual will be provided to participants.

COURSE 401

Friday

EDWARD B. SINGLETON, M.D.
Houston, Texas

Pediatric Chest Abnormalities

1. Technique
2. The normal chest and variations of normal
3. Abnormalities of the thoracic cage
4. Newborn pulmonary complications: a. variations of perinatal distress—hyalin membrane disease, aspiration syndrome and pulmonary hemorrhage; b. pneumonias including aspiration secondary to pharyngeal incoordination and esophageal atresia
5. Congenital anomalies: congenital lobar emphysema, congenital cystic malformation, diaphragmatic abnormalities, pulmonary agenesis and hypoplasia
6. Pulmonary abnormalities acquired in later infancy and childhood: a. types of pneumonia and their complications; b. acquired tracheoesophageal fistula; c. foreign bodies; d. fibrocystic disease; e. bronchiolitis
7. Rare pulmonary conditions: pulmonary alveolar microlithiasis, neoplastic diseases, pulmonary fibrosis, hemosiderosis, cytomegalic inclusion disease

COURSE 402

Friday

WILLIAM T. MESZAROS, M.D.
Chicago, Illinois

Roentgen Cardiology of Adults

1. The normal heart
2. Cardiac mensuration
3. Cardiac chamber enlargement
4. Hypertensive heart disease
5. Rheumatic heart disease
6. Arteriosclerotic heart disease
7. Luetic heart disease
8. Cor pulmonale
9. Miscellaneous disorders: a. thyroid heart disease; b. anemia; c. hemachromatosis; d. Addison's disease; e. dissecting aneurysm; f. scleroderma; g. carcinoid
10. Diseases of the pericardium
11. Congenital heart disease in adults
12. The lungs in heart disease

COURSE 403

Friday

BERTRAM R. GIRDANY, M.D.
Pittsburgh, Pennsylvania

Television Tape Studies of Fluoroscopic Procedures in Pediatric Radiology

The radiology department at the Children's Hospital of Pittsburgh utilizes television tape recording of the fluoroscopic image mediated through a newly developed image orthicon television camera optically coupled to a 9 inch image amplifier. The system permits recording on television tape and simultaneous viewing on the closed circuit television monitor at the same low levels of radiation required for closed circuit television viewing alone.

The television tape recordings are transferred to 16 mm. sound motion picture film for showing on standard projectors with or without sound. These recordings offer unusual opportunities for the study of all aspects of motion radiography.

Standard methods of fluoroscopic examination of infants and children with recordings of normal and abnormal conditions will be shown. Included are cardiospasm, congenital and acquired stenosis of the esophagus, H-type tracheoesophageal fistula without esophageal atresia, pyloric stenosis, palatal speech studies, neurogenic megacolon, malrotation, etc.

COURSE 404

Friday

ROBERT H. FREIBERGER, M.D.
New York, New York

The Roentgen Examination of the Foot

The interpretation of the roentgen examination of the foot with the exceptions of fractures and dislocations is very frequently delegated to the orthopedic surgeon. The course is designed to give the radiologist a basic knowledge of tarsal and forefoot alignment.

The following points will be covered:

1. Technique of roentgen-ray examination
2. An explanation of the terms varus and valgus and how they apply to the foot and ankle. Why the changes in tarsal alignment, primarily of the talus and calcaneus, can be expressed in terms of varus and valgus. A minimal amount of anatomy will be included
3. The normal foot
4. Specific foot deformities such as flatfoot, metatarsus adductus, metatarsus varus, talipes equinovarus, congenital plantar flexion of the talus, calcaneus and calcaneo-cavus
5. Recognition of tarsal coalitions

The course is designed to correlate physical and roentgen examination.

COURSE 407

Friday

LEO S. FIGIEL, M.D.
Detroit, Michigan

The Colon: Roentgenographic Considerations

The material presented in this course will establish evidences of a relationship between the colonic polyp and colonic cancer. The detection, significance and follow-up studies of selected case material dealing with the colonic polyp will be presented.

The incidence of error in the detection of colonic cancer will be discussed and measures to prevent such errors will be stressed.

The ileocecal region and the appendix will be covered in detail. The remainder of the course will deal with the barium enema examination in various obstructive conditions other than carcinoma, *e.g.*, volvulus, colo-colic intussusception and others. The more unusual entities in which a specific diagnosis is frequently possible such as endometriosis, pneumatosis coli, etc., will also be considered.

COURSE 410

Friday

KURT AMPLATZ, M.D.
Minneapolis, Minnesota

Techniques of Angiography

This course will deal with the various methods and techniques of angiography and to some extent with the indications and reasons for selecting one procedure over another. Peripheral arteriography, aortography, renal angiography, coronary arteriography, left ventriculography, carotid, vertebral and subclavian angiography will all be discussed. Percutaneous puncture with insertion of catheters of various types will be stressed. The desirability of using some type of pressure injector will be pointed out as well as many other facets of angiographic technique. Questions will be welcomed.

COURSE 421

Friday

JOSE SALA, M.D.
Columbia, Missouri

Treatment of Carcinoma of the Uterine Cervix

This course will be based on the experience at The Ellis Fischel State Cancer Hospital from 1943 to the present. Different techniques will be reviewed, including the use of transvaginal irradiation and curie therapy. Five year results will be given for the various methods.

COURSE 422

Friday

FRANK BATLEY, M.B., ChB., D.M.R.T.
Syracuse, New York

Common Errors in Radiotherapeutic Technique

The following philosophy of radiotherapy will be illustrated. Cobalt therapy cannot replace interstitial implantations for small mouth lesions, yet, gold grains are often preferable to radium needles. This heresy is held with the belief that only those implantations should be performed in which the dose can be calculated or nearly calculated. Large implants are advised as often as commando surgery.

The method of using parallel opposing fields with external irradiation is the easiest and worst technique. Less than three fields should never or hardly ever be used.

Radiosensitivity and radioresistance are now old-fashioned and misleading concepts, but the terms radiocurable and radio-incurable are not. One should know the limitations—or at least the limitations of the patient's tissues.

Illustrations will include reminiscences of the Holt Radium Institute in Manchester, consisting of the commencement of the "Find Point A Game," the use and abuse of the Paterson-Parker rules demonstrated by an electric toaster, the devaluation of the British roentgen, and the experiences of some early days with the first or second cobalt unit in Canada—2 half lives later.

COURSE 423

Friday

WALTER T. MURPHY, M.D.
Buffalo, New York

Complications of Head and Neck Irradiation

Radiation therapy of head and neck tissues is indicated for a great variety of primary and metastatic neoplasms. Sometimes such treatment precedes or follows surgical or chemotherapeutic procedures. Risks of cancerocidal irradiation are always real even with modern techniques. Avoidance of complications is made easier by an understanding of what can happen to patients irradiated for different clinical entities and with different radiotherapeutic techniques.

COURSE 424

Friday

JAMES J. NICKSON, M.D.
New York, New York

Applied Radiation Biology

In this course the fundamentals of radiation biology with particular reference to the clinical situation will be discussed. Consideration will be given to the opportunities that exist for improvement of the results of radiation therapy, and also to the opportunities that exist for advancing our knowledge in radiation biology by appropriately designed observations and trial studies in patients.

COURSE 425

Friday

FERNANDO G. BLOEDORN, M.D.
Baltimore, Maryland

The Combined Use of Radiotherapy and Surgery in Treatment of Cancers of the Lung and Esophagus

A program of combined radiotherapy and surgery for lung and esophageal carcinomata was established at the University of Maryland Hospitals in 1956.

This course will consider the basis for such an effort, the reasons for failure to cure these lesions, the philosophy and techniques of treatment, the complications of the method, and an analysis of results.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 26, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine Physics and Radiobiology	
101—Finby	Male Cystourethrography	**121—del Regato**	El Papel de la Radioterapia en el Tratamiento del Cancer (In Spanish)
102—Hope	Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-urinary Tract of Infants and Children	**122—Sanger**	Diagnostic Uses of Radioisotopes (Other Than for Thyroid Disease)
103—Morgan	Screen Intensification and Cineradiography	**123—Williams**	The r, rad and rem in Specification of Dose
104—Rubin	The Development of a Dynamic Classification of Bone Dysplasias	**124—D'Angio**	Radiation Therapy in the Management of Children with Tumors
105—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
106—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
107—Weens Gay	Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes		
108—Levin	The Fundamentals of Bone Disease		
109—Macmillan	Sinuses		
110—Nelson	Bronchography		
111—Wittenborg	Pelvis and Hips in Infancy and Early Childhood		
112—Egan	Over Five Years' Experience with Mammography		
113—Marshak	The Small Intestine		

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **September 27, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
201—Fischer Dodd Debbas	Lymphography	**221—Sheline**	The Use of Radioisotopes in the Diagnosis and Treatment of Thyroid Disease
202—Hope	Mediastinal Mass Lesions in Infants and Children	**222—Stein**	The Use of Megavoltage Therapy in the Treatment of Internal Cancer
203—Nice	Roentgenographic Manifestations of Collagen Diseases	**223—Kramer**	Radiotherapeutic Retreatment: To Do or Not To Do?
204—Jacobson	Malignant Bone Tumors—Primary and Secondary	**224—Martin**	The Treatment of Pulmonary Metastases with Roentgen-ray Therapy
205—Dotter (Same as 405)	The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department	**225—Marcial**	Cancer of the Tongue: Its Therapeutic Management
206—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
207—Felson	Some Fundamentals in Chest Roentgen Interpretation		
208—Levin	The Fundamentals of Bone Disease		
209—Macmillan	Mastoids, Lateral Neck and Larynx		
210—Nelson	Bronchography		
211—Schatzki	Differential Diagnosis of Lesions at the Lower End of the Esophagus		
212—Eyler Ormond	The Acute Abdomen		

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M. (except Course 305)** **September 28, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
301—Barden	Interpretation of Radiologic Signs of Abnormal Pulmonary Function	**321—Hale**	A Diagnostic Physics Laboratory Course for Radiology Residents
302—Hilbish Bartter	Roentgen Findings in Diseases with Abnormal Calcium Deposition	**322—Peters**	Indications for Radiotherapy during the Course of Hodgkin's Disease
303—McDonnel	The Radiation Hazard	**323—Murphy**	Carcinoma of the Vagina and Female Urethra
304—Jacobson	The Vaterian Segment: Normal and Abnormal	**324—Roswit**	Cancer Chemotherapy in Radiologic Practice
305*—Barth Bailey Buchanan Present Wachowski	Current Opinions from the American College of Radiology on Principles of Ethics and Economics		
306—Shehadi	Radiologic Study of the Biliary Tract		
307—Felson	Some Fundamentals in Chest Roentgen Interpretation		
308—Epstein	Myelographic Changes Associated with Spinal Osteophytosis		
309—Lester	Introduction to Angiocardiography		
310—Marshak	The Small Intestine and Colon		
311—Wohl Kirkpatrick	The Role of the Radiologist in Bone Tumor Diagnosis		
312—Campbell Klatte	Cineradiography. Part I.		
313—Mellins	Nonobstructive Acute Abdominal Disorders		

*** Course 305 will start at 1:30 P.M. and will be held in Napoleon Room No. 3.**

CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **September 29, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
401—Singleton	Pediatric Chest Abnormalities	**421—Sala**	Treatment of Carcinoma of the Uterine Cervix
402—Meszaros	Roentgen Cardiology of Adults	**422—Batley**	Common Errors in Radiotherapeutic Technique
403—Girdany	Television Tape Studies of Fluoroscopic Procedures in Pediatric Radiology	**423—Murphy**	Complications of Head and Neck Irradiation
		424—Nickson	Applied Radiation Biology
404—Freiberger	The Roentgen Examination of the Foot	**425—Bloedorn**	The Combined Use of Radiotherapy and Surgery in Treatment of Cancers of the Lung and Esophagus
405—Dotter (Same as 205)	The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department		
406—Shehadi	Radiologic Study of the Biliary Tract		
407—Figiel	The Colon: Roentgenographic Considerations		
408—Epstein	The Radiographic Investigation of the Basilar Cisterns		
409—Lester	Advanced Angiocardiography		
410—Amplatz	Techniques of Angiography		
411—Wohl Kirkpatrick	The Role of the Radiologist in Bone Tumor Diagnosis		
412—Campbell Klatte	Cineradiography. Part II.		

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

THE ATOM AND THE ENERGY REVOLUTION. By Norman Lansdell. Cloth. Price, $6.00. Pp. 200, with 32 illustrations. Philosophical Library, Inc., 15 East 40th Street, New York, N. Y., 1958.

This very readable book is addressed to the "general reader" who wishes to learn more about the social and political impact of the great atomic energy revolution. It should prove of interest to the men in business and industry, who must adapt themselves to the new economy, as well as to the specialist in related fields of inquiry. After a discussion of world resources of fuel and of expected world demands for increasing amounts of energy, the author describes various new sources of energy, notably, atomic energy. Fission and fusion energy and their releases are discussed in detail, followed by a description of the natural sources of materials for atomic energy developments. Interesting ideas are presented on radiation risks and insurance against them, on the influence of atomic energy development upon other industrial techniques, on the influence of atomic energy on the balance of world trade and finally on the relation of private industry to the state in respect to atomic energy. The wide scope of the impact on world affairs from the development of atomic energy is impressively presented. Unfortunately, the book has no index, which, in a work of this type, written for the general reader, would have been especially useful. A three-page glossary of scientific terms partially makes up for this omission.

OTTO GLASSER, PH.D.

BOOKS RECEIVED

THE SPINAL CORD; BASIC ASPECTS AND SURGICAL CONSIDERATIONS. By George Austin, Professor and Head, Division of Neurosurgery, University of Oregon Medical School; Chief Consultant in Neurosurgery, Veterans Administration Hospital, Portland, Oregon; Formerly, Assistant Professor of Neurosurgery, University of Pennsylvania. Cloth. Price, $26.50. Pp. 532, with 363 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

PROTECTION AGAINST RADIATION; A PRACTICAL HANDBOOK. By John D. Abbatt, M.B., Ch.B., D.M.R.; J.R.A. Lakey, Ph.D., B.Sc., A.Inst. P.; and D. J. Mathias. Cloth. Price, $6.50. Pp. 235, with 44 illustrations and 35 tables. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

CANCER OF THE NASOPHARYNX: ITS NATURAL HISTORY AND TREATMENT. By M. Lederman, M.B., D.M.R., F.F.R., Deputy Director, Radiotherapy Department, Royal Marsden Hospital; Consulting Radiotherapist, The Royal National Throat, Nose and Ear Hospital, The Metropolitan Ear, Nose and Throat Hospital, Moorfields Eye Hospital, The Royal Eye Hospital, The Chelsea Hospital for Women, London, England. Cloth. Price, $6.75. Pp. 117, with 53 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

PROBLEMS OF INFECTION, IMMUNITY AND ALLERGY IN ACUTE RADIATION DISEASES. From the Russian by N. N. Klemparskaya; O. G. Alekseyeva; R. V. Petrov; and V. F. Sosova. Translated by Lydia Venters, Argonne National Laboratory, Lemont, Illinois, Cloth. Price, $7.50. Pp. 165, with 34 illustrations and 29 tables. Pergamon Press, Inc., 122 East 55th Street, New York 22, N. Y., 1961.

LE NEOPLASIE MALIGNE DELLA TIROIDE; DIAGNOSI E TERAPIA. By Franco Chiariotti. Paper. Pp. 187, with many illustrations and tables. Minerva Medica, 1961.

ANNUAL REVIEW OF MEDICINE. Volume 12. David A. Rytand, Editor, Stanford University School of Medicine; and William P. Creger, Associate Editor, Stanford University School of Medicine. Cloth. Price, $7.00. Pp. 455, with few illustrations and tables. Annual Reviews, Inc., Palo Alto, California, 1961.

IXTH INTERNATIONAL CONGRESS OF RADIOLOGY. Volume I. Edited by Prof. B. Rajewsky, Dr. phil. nat., Dr. med. h. c., Dr. med. h. c., Frankfurt a. M., Präsident des Kongresses. Cloth. Pp. 800, with numerous illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U. S. A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

IXTH INTERNATIONAL CONGRESS OF RADIOLOGY. Volume II. Edited by Prof. B. Rajewsky, Dr. med. h. c., Dr. med. h. c., Frankfurt a. M., Präsident des Kongresses. Cloth. Pp. 825, with numer-

ous illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U. S. A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Acta Radiologica Supplementum 196. Mammary Carcinoma; the Biologic Character of Mammary Carcinoma Studied in 517 Cases by a New Form of Malignancy Grading. By K. A. Hultborn and Bo Törnberg. Paper. Price, Sw.Kr. 35:-. Pp. 143, with 74 illustrations and 140 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 197. The Mitotic and Radioprotective Effect of Cysteine and Lysine in Rat. By Lars R. Holsti. Paper. Price, Sw. Kr. 30:-. Pp. 99, with 17 illustrations and 18 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 198. The Isometric Relaxation Phase of the Left Ventricle; an Electrokymographic Study. By Osborne Bartley. Paper. Price, Sw. Kr. 35:-. Pp. 116, with 49 illustrations and 14 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 199. Vaso-Seminal Vesiculography in Hypertrophy and Carcinoma of the Prostate with Special Reference to the Ejaculatory Ducts; a Clinical and Experimental Roentgenological Study. By Gunnar Willer Vestby. Paper. Price, Sw. Kr. 35:-. Pp. 194, with 119 illustrations and 17 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 200. Contrast Examination of the Cardiovascular System during Increased Intrabronchial Pressure. By Björn Nordenström. Paper. Price, Sw. Kr. 30:-. Pp. 110, with 51 illustrations and 1 table. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplement No. 201. Risa Encephalography and Conventional Neuroradiologic Methods; a Comparative Study. By Giovanni Di Chiro. Paper. Price, Sw. Kr. 35:-. Pp. 102, with 38 illustrations, 60 sketches and 3 diagrams. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1961.

Therapeutic Dose Distributions with High-Energy Radiation. Survey of Existing Data and Report of a Panel on Physical Data for Dose Distributions with High-Energy Radiation Held at Vienna, 7–11 November 1960. Paper. Pp. 72. International Atomic Energy Agency, Kaerntnerring 11, Vienna 1, Austria, 1961.

VOL. 86, NO. 2

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. LEUCUTIA, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Skeletal System

Hunt, James Cleon, and Pugh, David G. Skeletal lesions in neurofibromatosis. *Radiology*, Jan., 1961, *76*, 1–20 (Address: J. C. Hunt, Mayo Clinic, Rochester, Minn.)

Neurofibromatosis (Recklinghausen's disease) is a congenital and familial disease presenting abnormalities of the skin, nervous system, bones and soft tissues. While the neurectodermal tissues have been accepted as being the tissues more commonly involved, recent studies have shown that mesodermal dysplasia is also a basic component, accounting for most of the skeletal defects found in this disease.

A total of 192 cases in which a positive diagnosis of neurofibromatosis was made during a ten year period from 1948 through 1957 at the Mayo Clinic was studied for skeletal defects. In 97 (51 per cent) some skeletal abnormality was demonstrated roentgenographically. These changes, when analyzed, classified and tabulated, revealed that skeletal defects of a type considered to be characteristic were present in 74 cases (39 per cent) and should suggest the possibility of this disease to the radiologist.

These defects include: severe angular scoliosis with dysplasia of vertebral bodies (14 cases); defects of the posterosuperior wall of the orbit (13 cases); disorders of bone growth associated with elephantoid hypertrophy of overlying soft tissue (18 cases); congenital bowing and pseudoarthrosis (4 cases); erosive defects of bone from contiguous neurogenic tumors (36 cases); and intrathoracic meningocele (1 unproved case).

Other skeletal abnormalities, not characteristic but possibly representing mesodermal dysplasia, consisted of congenital anomalies of many types and a mild scoliosis. Thirty-three examples were found of the former and 14 of the latter. Intraosseous cystic lesions, described by some writers as a characteristic manifestation of neurofibromatosis, were not thought by the authors to fall in this classification. No instances of this lesion were found in this series of cases. Osteomalacia, considered by some to be an incidental finding in neurofibromatosis, was also not found.

The incidence of neurofibromatosis among admissions to the Mayo Clinic during the ten year period was 1 in 3,100, and there were 9 cases (5 per cent) in which sarcomatous degeneration occurred.—*D. N. Dysart, M.D.*

Retz, Louis D., Jr. Primary liposarcoma of bone: report of a case and review of the literature. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 123–129. (From: Department of Surgery, State University of New York, Upstate Medical Center, and the Surgical Service, Veterans Administration Hospital, Syracuse, N. Y.)

A case is reported of a liposarcoma confined to the medullary cavity of the tibia. A forty year old male had a single well circumscribed central lytic defect in the upper metaphysis of the tibia measuring 4 by 5 cm. in size. The epiphyseal plate was intact. There was no periosteal reaction or cortical erosion. There was loss of the normal trabecular pattern and a "ground glass" appearance.

The lesion was curetted and packed with bone but four months later there was extension down the shaft. A midthigh amputation was done and two years later there was no evidence of recurrence or metastases.

Pathologic examination showed no tumor outside the bone and no cortical breakthrough. The tumor was well differentiated, with areas of typical nonossifying fibroma associated with sarcoma and an abundance of intracellular lipids. There were spindle shaped foamy lipoblasts and giant vacuolated sarcomatous cells. Considerable lymphocytic infiltration was seen but no necrosis.

Liposarcomas are found most frequently in the thigh, popliteal space, gluteal region and retroperitoneal space. Only 7 cases of intramedullary lipoma and 14 cases of liposarcoma of bone have been reported. None of the latter were confined solely to the medullary cavity, as in the present case. The more differentiated liposarcomas are slow growing and usually do not metastasize, but they may recur if they are not adequately excised. The less differentiated tumors have a poor prognosis and metastasize through the blood stream, commonly to the lungs, other bones and the brain.—*Martha Mottram, M.D.*

Scaglietti, O., and Stringa, G. Myxoma of bone in childhood. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 67–80. (From: Istituto Ortopedico Toscano dell'Università di Firenze, Florence, Italy.)

This rare bone tumor closely resembles chondromyxoid fibroma but always shows recurrence after simple curettage; therefore, initial resection is indicated when the diagnosis is established.

Two new cases are presented as well as 3 previously reported, all of which developed during the first decade. There was rapid development of pain, swelling and limitation of motion.

The lesions were visualized roentgenographically as irregularly rounded, lobulated, eccentric radiolucent defects in the metaphyses and showed a marked increase in the thickness of the cortical bone and diffuse sclerosis of the underlying spongiosa. Grossly, they resembled gelatinous cysts with septa. The tumor appears to arise in the subperiosteal extracortical region and penetrates the spongiosa. Each lobule of the tumor contained elongated stellate or triangular cells with a margin of closely packed round cells and foreign body giant cells. The intercellular substance had a uniform thin fibrillar appearance with metachromatic staining. An older myxoma may

develop fibromatous elements, so it may be termed fibromyxoma.

Although some might classify myxomas as juvenile forms of chondromyxoid fibroma because they appear so similar, it is urged that myxomas be considered in a separate category since they occur before the age of ten and are characterized by marked pain and swelling with rapid progression, marked periosteal reaction seen on roentgenograms, predominance of myxoid tissue and a positive reaction for metachromasia in the ground substance, and invariable recurrence after simple curettage but complete recovery after resection.—*Martha Mottram, M.D.*

Schajowicz, Fritz. Giant-cell tumors of bone (osteoclastoma); a pathological and histochemical study. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 1–29. (From: Center of Osteoarticular Pathology and the Department of Orthopaedic Surgery and Traumatology, Italian Hospital, Buenos Aires, Argentina.)

This is a review of 85 giant cell tumors with 41 complete specimens available, so that histochemical studies and tissue cultures could be done. Multiple histochemical studies were carried out in giant cell tumors as well as in other bone and soft tissue lesions which contained giant cells. For details as to types of enzymes, carbohydrates and lipids tested, the reader is referred to the original paper.

The author believes from his pathologic studies that the giant cell is of mesenchymal origin and is formed by agglutination and conglomeration of the mononuclear cell as well as by repeated division of the stromal cell nuclei by amitosis or nuclear segmentation. He feels that the giant cell tumor is a neoplastic derivation of the osteoclast.

Giant cells have an abundance of acid phosphatase, betaglucuronidase, succinic-dehydrogenase and no alkaline phosphatase. There are also complex lipids, probably glyco- or phospholipids. The stromal cells of giant cell tumors generally show no enzymatic reactions, nor do they possess glycogen, mucopolysaccharides or glycolipids in significant amounts.

The histochemical findings are identical in the multinucleated giant cells of giant cell tumors and the so-called variants and in the normal chondroclastic and osteoclastic giant cells.

The author concludes that histochemical study is of no diagnostic value in differentiating giant cell tumor from its variants or in defining prognosis. In the exceptional case with rapid evolution and recurrence, differentiation from osteolytic osteogenic sarcoma can be made easily by the presence of intense alkaline phosphatase activity in the latter. The histologic, clinical and roentgenographic findings could not be correlated sufficiently to grade the giant cell tumors.

Radical resection of the tumor with bone grafts is recommended because of an increasing number of cases of benign or agressive giant cell tumors which have produced metastases with identical histologic patterns and which showed progressive growth, recurred or transformed into sarcoma.—*Martha Mottram, M.D.*

Bose, K. S. Observations on changes of pattern in chronic pyogenic osteomyelitis following inadequate administration of penicillin: their similarity with other bone diseases. *J. Indian M. A.*, Apr., 1959, *32*, 271–273. (From: Fracture and Orthopaedic Department, Medical College, Calcutta, India.)

This is a report on 7 cases of chronic pyogenic osteomyelitis which had been inadequately treated with antibiotics and which presented atypical features simulating various lesions of bone, but excluding malignancy. These cases were investigated from clinical, roentgenologic and histologic aspects, with an attempt to categorize the lesions insofar as possible. A definitive diagnosis on the basis of the clinical and roentgen findings is often difficult and may depend on an adequate biopsy specimen and evaluation of the clinical course over a period of time.

It was found that inadequate antibiotic administration in the acute stage of pyogenic osteomyelitis resulted in unchecked virulence of the organism, more marked bony necrosis and rarefied areas which roentgenographically simulated bone diseases such as hemangioma, fibrocystic disease and Gaucher's disease. Moreover, in other case, new bone formation was seen to be more marked, simulating sclerosing bone diseases such as osteoid osteoma, Paget's disease, infantile cortical hyperostosis, and Engelman's disease.—*Raymond V. Hussey, Captain, USAF MC*

Bose, K. S. Atypical osteomyelitis following inadequate antibiotic therapy: its similarity with fibrous dysplasia. *J. Indian M. A.*, Dec., 1959, *33*, 464–468. (From: Department of Surgery, Medical College Hospitals, Calcutta, India.)

Four cases of chronic pyogenic osteomyelitis following inadequate penicillin treatment are presented with an analysis of the clinical features, roentgen findings, histology, treatment and results.

The roentgen changes included minimal bone necrosis with absence of sequestra and little new bone formation. Also, there was a slowly developing cystic osteoporosis involving the diaphysis with expansion of the shaft and an altered trabecular pattern usually associated with fibrous dysplasia.

The striking feature of this investigation was that 2 of the 4 cases showed typical histologic and roentgen features of fibrous dysplasia but positive biopsy cultures revealed organisms of *Streptococcus viridans* and *Bacillus fecalis alcaligenes.*

The author concludes that infection may well be one factor in the etiology and pathogenesis of fibrous dysplasia.—*Raymond V. Hussey, Captain, USAF MC*

Sutro, C. J., Ehrlich, D. E., and Witten, M. Low back disorders; values of radiographs of the dorsal vertebrae in evaluating the etiology of lumbosacral derangements. *Bull. Hosp. Joint Dis.*, Apr., 1960, *21*, 42–49. (From: Brooklyn Outpatient clinic, U. S. Veterans Administration Hospital, Brooklyn, N. Y.)

Patients with severe pains in the lumbar area or in the sacroiliac region may, on roentgen examination, present minimal or negative findings in the soft or osseous parts of the lumbar vertebrae or of the pelvic bones.

It should be remembered that the lower dorsal nerves innervate the skin in the lumbar, sacroiliac and femoral trochanteric regions. Thus, lesions situated in the lower dorsal vertebrae or in their contiguous soft parts may cause referred pains in the lumbar or pelvic regions.

The authors present 3 cases of such referred pain illustrating the shortcomings of segmental roentgen examination and emphasizing the necessity of including roentgen examination of the dorsal vertebrae in addition to that of the lumbar and pelvic bones in cases of low back disorders.—*Charles H. Helmen, M.D.*

Vyhnánek, Luboš, Lavička, Josef, and Blahoš, Jaroslav. Roentgenological findings in gout. *Radiol. Clin.*, July, 1960, *29*, 256–264. (From: The Radiological Clinic of the Charles-University in Prague, and the Clinic of Internal Medicine in Pilsen, Czechoslovakia.)

Six per cent of the patients with chronic inflammatory rheumatism were found to be suffering from gout. The authors describe the roentgen changes in gout based on their experience with 44 patients.

These changes are divided into three groups: in Group I (20 patients) the findings were negative or nonspecific, in Group II (11 patients) there was localized osteoporosis of the joint and subchondral cystoid areas without damage to the bony cortex; and in Group III (7 patients) there were cystoid areas with damage to the bone cortex and destruction of the joint.—*James J. McCort, M.D.*

Blood and Lymph System

Sheehan, R., Hreshchyshyn, M., Lin, R. K., and Lessmann, F. P. The use of lymphography as a diagnostic method. *Radiology*, Jan., 1961, *76*, 47–53. (Address: R. Sheehan, Roswell Park Memorial Institute, Buffalo 3, N. Y.)

The lymphatic system has been visualized roentgenographically by a number of investigators using the injection of opaque substances, but the first real clinical application was not made until 1952.

The authors report their experiences with lymphangiography undertaken as a diagnostic method in 40 patients with suspected disease of the pelvic or retroperitoneal lymph nodes. The lymphatics were first outlined with direct sky blue or Evans blue dye injected subcutaneously into the web of the foot between the first and second phalanges. A cut down was then made on the dorsum of the foot; the lymph vessel was located; and the contrast medium was injected. In 7 cases, a water soluble medium was used, with only 3 satisfactory results. An oily medium was employed in 31 studies with satisfactory diagnostic results in 27 of these. Ethiodol, which contains 37 per cent iodine in organic combination with the ethyl esters of the fatty acid of poppyseed oil, seemed to produce the best results.

In general, the lymph nodes were visualized up to the level of L-2. Complications were minimal, with gradual absorption of the contrast medium. The procedure seemed to have greater diagnostic value in lymphomas than in metastatic lymph node involvement. Small metastatic lesions were not detectable. The lymph nodes completely filled with tumor did not take up the dye, but displacement of the neighboring channels was sometimes shown. Incomplete filling or inflammatory changes occasionally caused defects which were difficult to interpret.

Venography or intravenous pyelography can visualize pathologic lesions in many cases, while lymphography can be of value in cases where lymphogenous involvement cannot be determined by conventional methods. Lymphography can also be used to explore the normal lymphatic system and to evaluate changes following chemotherapy. The procedure is time consuming and experience is needed for correct interpretation of the results.—*James C. Moore, M.D.*

RADIATION THERAPY

Arneson, A. N., and Williams, Carol F. Long-term follow-up observations in cervical cancer. *Am. J. Obst. & Gynec.*, Oct., 1960, *80*, 775-790. (From: Department of Obstetrics and Gynecology, and the Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo.)

The authors report a statistical study of their long term follow-up observations in cervical cancer. A consecutive series of 394 patients seen between the years 1935 and 1954 was reviewed. Of this group, there were 374 patients with invasive cancers who received treatment: 32 per cent were Stage I, 48.5

per cent Stage II, 16.5 per cent Stage III, and 3 per cent Stage IV. The five year survival for Stage I cases was 78.3 per cent, for Stage II 54.1 per cent, and for Stage III 37.1 per cent. There were no Stage IV five year survivals. The five year over-all survival rate was 57.5 per cent. The five year survival values give a realistic view of cure rates. A patient clinically well after five years has little probability of dying from cancer.

A five to twenty year follow-up is also reported for this series of patients. To test the effectiveness of treatment over the years, the series is divided into two groups of approximately equal numbers—those seen before and those after 1949. The relative five year results for these two groups are compared. An increase in survival is not demonstrated in the more recently treated patients, despite the fact that the percentage distribution of early lesions increased. This difference in results is then studied by using cumulative survival rates.

The authors' experience with programs of secondary irradiation has been unfavorable in the treatment of persistent or reappearing cancer. In more than 50 such attempts, only 2 patients have survived an additional five years after a second course of radiation therapy.

The rate of major complications, such as the incidence of fistula formation, has fallen from 11 per cent in the earliest years to 2 per cent in the 1950–1954 period. This improvement is taken as evidence of an advance in treatment technique, despite the lack of increased survival rates for the most recent years.—*Richard L. Jontz, M.D.*

Sotto, Luciano S. J., Graham, John B., and Pickren, John W. Postmortem findings in cancer of the cervix; an analysis of 108 autopsies in the past 5 years. *Am. J. Obst. & Gynec.*, Oct., 1960, *80*, 791–794. (From: Departments of Gynecology and Pathology, Roswell Park Memorial Institute, Buffalo, N. Y.)

From June, 1954 to May, 1959, a total of 108 autopsies were performed at Roswell Park Memorial Institute on patients diagnosed and treated previously for carcinoma of the uterine cervix. Of these patients 85 per cent had been treated during the preceding five years; 75 per cent had received their primary treatment at the Institute; and 90 per cent of the total had received what is now considered to be adequate treatment.

In 23 per cent, the original stage of disease was unknown because of initial treatment elsewhere; 21 per cent were Stage I, 23 per cent Stage II, 21 per cent Stage III, and 12 per cent Stage IV.

Regarding the clinical response to primary treatment, 37 per cent had persistent pelvic disease, 52 per cent recurrent pelvic disease, 9 per cent no pelvic tumor, and 2 per cent unknown.

The leading cause of death was renal failure (37 per cent), manifested by uremia due to obstruction or pyelonephritis.

Of the 93 patients having tumor present at autopsy, in only 15 per cent was the disease limited to the pelvis. In 28 patients there was bladder involvement and in only 12 was the rectum invaded.

Distant metastases were found in 79 patients (85 per cent) with the lung (38 per cent) and liver (37 per cent) being the most common sites; 20 per cent of the cases had bone involvement, mainly in the spine.

Lymph node metastases were found in 61 of the 93 patients having disease; in 6 instances these were localized in the pelvis; in 33 they were extrapelvic; and in 22 they were both pelvic and extrapelvic.—*R. L. Turner, M.D*

Sheline, Glenn E., Goldberg, Minnie B., and Feldman, Robert. Pituitary irradiation for acromegaly. *Radiology*, Jan., 1961, *76*, 70–75. (Address: G. E. Sheline, Department of Radiology, University of California Medical Center, San Francisco 22, Calif.)

This paper presents the experience gained between January, 1942 and March, 1959 at the University of California Hospitals, San Francisco, with radiation therapy for acromegaly in 37 patients. During this time the diagnosis of acromegaly was made in a total of 65 patients. Seven cases were discarded because of inadequate information, 9 were considered quiescent, 2 patients had hypophysectomy and 1 was treated by estrogen therapy alone. Nine other patients were excluded for various reasons.

In this series, both the "interval" and the "single course" techniques were employed. Ten patients received a calculated pituitary dose of 1,000 to 2,500 r. In the other 27 patients, the dose for the initial course of treatment was 2,550 to 6,000 r, usually given in a period of four to eight weeks. Subsequently, several of those given doses between 2,550 and 3,500 r received additional therapy. External roentgen rays (200, 400 or 1,000 kvp.) were used with multiple stationary fields or a rotational technique. Therapy was usually begun with a daily pituitary dose of 50 r or less and gradually increased to an average of about 700 r per week. The daily dose was determined by the patient's tolerance. The results of irradiation were gauged according to the response evident at six weeks, three months, and twelve months following completion of each course of therapy. The follow-up period ranged from one to eighteen years.

Twenty-eight of the 37 patients improved following the initial course of roentgen therapy. In 19 the disease was permanently controlled. The improvement was either incomplete or transient in 9. A dose in excess of 3,500 r resulted in improvement in 78 per cent of the cases, whereas a lower dose brought the disease under control in only 26 per cent; the latter

figure was raised to 63 per cent by retreatment. The disease was considered to be quiescent if headache, progression of visual field impairment, sellar destruction, skeletal growth, soft tissue swelling and other evidence of endocrine hyperactivity ceased. The visual fields became normal in 10 of the 14 patients for whom data are available.

These results seem to confirm the contention that visual field defects in acromegaly do not constitute a contraindication to pituitary irradiation unless they are recent, acute, and rapidly progressive. No significant complication developed in any patient receiving a single course of treatment, even with tumor doses up to 6,000 r.—*A. W. Sommer, M.D.*

CHIARIOTTI, FRANCO. Alcuni singolari aspetti delle neoplasie tiroidee. (Unusual aspects of thyroid neoplasms.) *Radiol. med.*, Aug., 1960, *46*, 738–756. (From: Ospedale di Circolo di Busto Arsizio, Italy.)

In a broad sense thyroid cancer is characterized by "unusual aspects" from the anatomic, pathologic, clinical, diagnostic and therapeutic standpoints. The observations which are presented are the result of the study of 164 cases of thyroid tumors treated in the hospital by different methods.

(1) *Discrepancies between the histologic characteristics and the clinical course of thyroid tumors.* The author notes the long survival of patients with diffuse visceral and skeletal metastases and observes that tumor types that might be classified as benign cytologically, may be the origin of extensive generalized metastases (benign metastasizing struma, benign metastasizing thyroid adenoma). He surveys the contributions of other investigators and cites pertinent cases studied by him. Most interesting among these is that of a woman thirty-six years of age who presented a diffuse nodular metastatic dissemination in both lungs which had remained stationary for thirteen years with no appreciable complaints. The functional thyroid origin of the metastases was established by scanning following the administration of I^{131}.

(2) *Malignant tumor in aberrant thyroids.* Topographically one may describe aberrant thyroids as median and lateral. The median which are derivations of the thyroglossal duct have been designated as lingual. Because of the frequency of the malignancy in these sites, prompt determination of the nature of the lesion and therapeutic intervention are indicated. It is difficult to differentiate between cervical lymph node metastases and lateral aberrant thyroids.

(3) *Malignant thyroid tumors in the young.* A survey of the cases reported in the literature and of those included in this study apparently points to an increase in the incidence of malignant tumors observed in the first decades of life. The author notes that, in 4 of the 12 cases he studied, the first symptom was the appearance of lateral cervical tumefactions. Juvenile thyroid carcinomata react favorably both in the primary site and in metastases, especially when surgical intervention is combined with internal and external radiation therapy. He reports an average survival of six to twelve years in subjects still alive.

(4) *Goiter and thyroid cancer.* The incidence of thyroid cancer in previously existent goiter, as reported by the author and by other contributors quoted by him, is between 20 per cent and 50 per cent.

(5) *The relationship of previous irradiation of the neck and the appearance of malignant thyroid neoplasm.* The author, on the basis of the cases reported in the literature, states that radiation therapy of the neck and mediastinum is assumed to favor development of cancer in later years. His series of 12 cases of juvenile thyroid cancer includes only 1 which had received previous radiation therapy to the neck, and does not confirm this assumption.

(6) *Diagnosis of thyroid neoplasm.* The diagnosis is based principally on clinical examination and must be confirmed by biopsy. Some authors insist on the necessity of serial histologic examinations. Others control the extent of their operative intervention by frozen sections. The difficulties of the histologic diagnosis are due to the coexistence of diverse cytologic types in the same specimen, be it from a primary or a metastatic site. Roentgen examination is of great aid in establishing the diagnosis. It includes conventional roentgenography, laminagraphy, arteriography, pneumothyreography, and pneumomediastinography. It will aid in the recognition of the location and extent of the primary and secondary lesions, visceral and skeletal. The scanning of the thyroid region after the administration of I^{131} usually shows a diminished uptake at the site of malignancy. Scintiscanning of the body following proper preparation will outline metastatic foci, which have an appreciable uptake.

(7) *Therapy.* Radical surgical extirpation of the malignant thyroid, when operable, is the method of choice. It is to be followed by a period of treatment with radioiodine. Of 22 patients so treated by the author, 7 are living more than five years. In inoperable cases, in postoperative recurrences, and in metastases, the choice of external irradiation, isotope therapy, or a combination of both must be made. Each case is to be studied individually. When an appreciable uptake of radioiodine has been determined, treatment with the isotope is advisable. When the radioiodine is poorly concentrated, the primary lesion is treated by external irradiation. The metastases are then attacked by massive doses of radioiodine, administered at intervals, when they have acquired functional characteristics.

The author proposes a standardization of the classification of the various neoplastic histologic types, a universal definition of the stages of the

disease and uniformity in the dosage of radiation.—*Anthony A. Blasi, M.D.*

Barr, Nathaniel F., Stark, Mary, and Laughlin, J. S. A comparison of two fluorometers designed to measure the radiation-induced fluorescence of silver-activated glass rods. *Radiology*, Jan., 1961, *76*, 113–115. (Address: N. F. Barr, 410 E. 68th Street, New York 21, N. Y.)

The use of silver-activated phosphate glass in the form of small cylindrical rods (1×6 mm.) for dosimetry in radiobiologic, clinical, and health physics studies has led to the design of a variety of instruments for measuring the radiation-induced fluorescence. In a comparison of the results obtained with a commercially available Bausch and Lomb microdosimeter reader and a system employing Photovolt Model #540 fluorometer, it was found that the Bausch and Lomb instrument is ultimately 100 times more sensitive than the Sloan-Kettering system; however, because of the lack of uniformity (particularly in respect to the diameter) in the rods which are presently available, advantage cannot be taken of this extra sensitivity. The greater inherent sensitivity of the Bausch and Lomb reader would be of extreme importance if rods containing less silver were developed, since this would lessen their quality dependence.—*Walter H. Jarvis, Jr., M.D.*

RADIOISOTOPES

Manaro, Julio Morato, Paseyro, Pedro, and Grosso, Osvaldo F. El gammagrama y el citograma en el estudio del cancer de la glandula tiroides. (The gammagram and the cytogram in the study of thyroid carcinoma.) *An. Fac. de med. de Montevideo*, Mar.–Apr., 1960, *45*, 215–222. (From: Instituto y Clínica Endocrinológica del Ministerio de Salud Pública y Facultad de Medicina, Montevideo, Uruguay.)

Gammagrams may provide useful information in the diagnosis of thyroid tumors by showing the relationship of the thyroid gland to soft tissue masses and adjacent structures. In 8 case of thyroid gland carcinoma, 6 showed increased I^{131} uptake within forty-eight hours. This was attributed to the fact that in thyroid neoplasms seen in an early stage there was hyperplasia of that portion of the thyroid not involved by the tumor, resulting in an increased uptake of I^{131} without a rise in thyroid hormone secretion. Two cases showed uptake readings within normal limits. One of these, an adenocarcinoma, was considered to be too small to affect the thyroid function. Aspiration cytologic diagnosis was recommended as a safe simple technique which may provide valuable information of cell structure in blastomatous growths and related conditions.—*Murray M. Friedman, M.D.*

Abt, Arthur F., and Balkus, Vincent A. The radio-renogram, with radio-renografin-I^{131}, as a diagnostic aid in urologic problems. *J. Urol.*, Jan., 1961, *85*, 95–102. (From: Radioisotope and Genitourinary Services, Veterans Administration Center, Martinsburg, W. Va.)

Renography using I^{131} offers an additional test of renal function. It is particularly useful for evaluating the differential function of the two kidneys and, in conjunction with the other methods for evaluating the urinary tract, it is an addition to the armamentarium for renal function studies. This test compares favorably with conventional renal function tests, but it should not be considered to be in itself definitively diagnostic, as it can only indicate disparity in function and not exact etiologic conditions.

The authors have successfully used this test to evaluate function in patients with unilateral renal obstruction caused by calculus, tumor, or infection. It is also of some value in the detection of unilateral renal disease in association with hypertension and with the hypoplastic or absent kidney.—*George W. Chamberlin, M.D.*

Winter, Chester C., Nordyke, Robert A., and Tubis, Manuel. Clinical experience with a new test agent for the radioisotope renogram: sodium ortho-iodohippurate-I^{131} (hippuran-I^{131}). *J. Urol.*, Jan., 1961, *85*, 92–94. (From: Departments of Surgery [Division of Urology], Medicine, and Radiology, University of California Medical Center, Los Angeles, and the Radioisotope Service, Veterans Administration Center, Los Angeles, Calif.)

The authors point out that I^{131} seems to be the best available isotope to incorporate with intravenous radiopaque material for the radioisotope renogram. Diodrast I^{131} has the disadvantage of being secreted by the liver as well as the kidneys. The use of this drug sometimes necessitates large doses to block the liver excretion; furthermore, when using diodrast I^{131} it is necessary to angle the probes away from the liver, particularly on the right side. Urokon I^{131}, hypaque, or miokon I^{131} were found to be useful but their clearance from the blood is not as rapid as that of diodrast I^{131}.

Recently, Nordyke, Tubis and their associates found that hippuran I^{131} has a much more rapid and higher secondary rise and a more rapid descent on the tracing than does diodrast or the other media. This medium is stable, heat resistant, easily manufactured, commercially available, nontoxic, and has a biologic half life of 20 minutes. Urine assays in the

normal patient reveal as high as 75 per cent recovery of hippuran I^{131} in 30 minutes. The tests can be completed in two-thirds of the time required for hypaque.

The authors recommend hippuran I^{131} for renography at the present time.—*George W. Chamberlin, M.D.*

SIURALA, M., LAMBERG, B.-A., TURULA, K., and HARJANNE, A. Studies with I^{131}-labelled fat. IV. Absorption, distribution and excretion of radioactivity after ingestion of radioactive triolein. *Scandinav. J. Clin. & Lab. Invest.*, Jan., 1960, *12*, 97–106. (From: The Medical Department, Maria Hospital and the IVth Department of Medicine, University of Helsinki, Helsinki, Finland.)

The authors have measured plasma, urine, and fecal concentrations of I^{131} following the ingestion of radioactive triolein in 8 patients, 3 of whom had steatorrhea. In the normals, after eight hours, only 11 per cent of the ingested dose was not accounted for and total recovery eventually approached 100 per cent. Results show that most of the plasma I^{131} is contained in the nonprecipitated portion (20 per cent trichloroacetic acid was used for precipitation), and that total plasma radioactivity as it represents the iodine diffusion space is of greater practical significance than the plasma lipid bound radioactivity (precipitated portion). Total plasma recovery ranged from 47 to 63 per cent in the normals at six hours, but only 14 to 32 per cent in the patients with steatorrhea. It is also suggested that measurement of the radioactivity in the urine during the first four to six days may be a useful test for determining the amount absorbed, as the per cent recovered in normals approached 100 per cent but was much less in the abnormals.—*William K. Littman, M.D.*

LAMBERG, B.-A., SIURALA, M., TURULA, K., HAKKILA, R., and HARJANNE, A. Studies with I^{131}-labelled fat. V. Distribution of radioactivity after intravenous injection of radioactive triolein emulsion. *Scandinav. J. Clin. & Lab. Invest.*, Jan., 1960, *12*, 107–115. (From: The Medical Department, Maria Hospital and the IVth Department of Medicine, University of Helsinki, Helsinki, Finland.)

This report is a continuation of the authors' previous study of oral ingestion of radioactive triolein. However, this time the triolein was given intravenously and the circulating I^{131} was studied. About 97 per cent of the lipid bound radioactivity was shown to be removed from the blood in thirty minutes. This study again confirms that measurement of only the lipid bound I^{131} in plasma after oral or intravenous administration of labelled fat does not give reliable measurement of the amount absorbed or of distribution within the body. The most important fraction is the total plasma I^{131}, which is in accordance with the results of the authors' previous studies.—*William K. Littman, M.D.*

RUFFIN, JULIAN M., and TYOR, MALCOLM P. Radioisotopes as diagnostic aids in gastroenterology; an evaluation. *J.A.M.A.*, Feb. 25, 1961, *175*, 685–688. (From: The Department of Medicine, Duke University Medical Center and the Veterans Administration Hospital, Durham, N. C.)

I^{131} labeled lipids have been used in the study of intestinal absorption since 1949. At the present time, I^{131} labeled triolein and oleic acid are the most widely employed. Oleic acid is readily absorbed by the normal intestine, whereas triolein must be partially hydrolyzed before absorption can take place. Thus, by using the above materials one can differentiate a defect in digestion from an impairment in absorption.

Blood and stool samples are studied for their radioactivity and the results are considered to be normal if the average of the fourth, fifth and sixth hour blood samples is more than 10 per cent of the administered dose and if less than 5 per cent of the administered dose is present in the 48 to 72 hour stool specimens. Organic disease may exist even if the triolein studies are normal but the reverse is not true. The I^{131} labeled fat absorption studies are of value in steatorrhea, disease of the pancreas, sprue, evaluation of treatment of sprue and in differentiating diarrhea due to an irritable colon from that due to organic disease.

I^{131} labeled human serum albumin has been used to study protein absorption. However, proteins not undergoing normal digestion and absorption are decomposed by bacterial action resulting in abnormal amounts of indol and phenol derivatives in the blood and urine. I^{131} labeled polyvinylpyrrolidone has been used recently, since it does not undergo enzymatic action and, otherwise, reacts similarly to albumin. It has been used in studying idiopathic hypoproteinemia and also for evaluating protein loss in regional enteritis, ulcerative colitis and hypertrophic gastritis.

Cr^{51} labeled red blood cells have been used to study gastrointestinal bleeding. The patient's own red blood cells are tagged and injected intravenously; the stools are collected for 24 hour periods and, by comparing the radioactivity in the stools with that in the blood, the daily blood loss can be determined. The site of bleeding can also be determined by obtaining samples at different levels by means of gastrointestinal intubation. The Cr^{51} test is believed to be more specific and more sensitive than the chemical measurements currently employed.

Co^{60} labeled vitamin B_{12} has been used to study pernicious anemia, sprue and the blind loop syndrome. The absorption of Co^{60} labeled vitamin B_{12} is impaired in sprue and is not improved by the addition of either an intrinsic factor or treatment with

antibiotics. There is malabsorption of Co^{60} labeled vitamin B_{12} in pernicious anemia, which is corrected by the addition of an intrinsic factor. The absorption of Co^{60} labeled vitamin B_{12} is impaired in the blind loop syndrome (diverticulosis, blind loops, or strictures with secondary stasis and obstruction) and returns to normal following the addition of an antibiotic.

The above radioisotopes have been found to be useful in studying various aspects of digestion. Some of the tests do not correlate too closely with the chemical quantitative tests, but this does not detract from their usefulness. New uses of the present materials and the development of new materials in the study of digestion are to be anticipated.—*Charles W. Cooley, M.D.*

CORDREY, LEE J., CHRISTIE, JAMES H., MACINTYRE, WILLIAM J., and PALMER, RUFUS. A study of strontium 85 metabolism in the normal and in the castrated mouse. *J. Bone & Joint Surg.*, Dec., 1960, *42-A*, 1349–1362. (From: The Radioisotope Laboratory, Highland View Hospital, Cleveland, Ohio.)

The purpose of this study was to determine in mice the effect of castration on bone. Radioactive strontium 85 (Sr^{85}) was used because of its close similarity to calcium regarding its biologic behavior and especially regarding bone metabolism. Radioactive isotope evaluation was used because continuous results can be quantitated by this method. Sr^{85} was administered intraperitoneally to obviate the difference between calcium and strontium absorption from the intestinal tract.

The mice were injected with 10 μc of $Sr^{85}Cl_2$, and a similar amount was injected into a serum bottle filled with distilled water (approximately the size of a mouse). The radioactivity of each mouse was then counted (in a small cardboard box to determine the exact amount injected) and this was compared with the standard and normalized to 100 per cent; the subsequent disappearance of Sr^{85} from the mice was determined on this basis.

The effect of castration on some of the chemical values of bone was determined first, and it was shown that the calcium values in castrated mice were markedly elevated in the period immediately following castration and then fell off progressively with time; after five weeks the values were uniformly below the normal range. The biologic turnover of Sr^{85} was determined on a group of mice and it was found that there were three phases: (1) an initial relatively rapid turnover which lasted for forty-eight days, (2) a slower decrease in excretion, and (3) a very slight release of Sr^{85}. The distribution of Sr^{85} in the mouse was studied and, five days after the injection of Sr^{85}, 99 per cent was found to be retained in the skeleton. It was also determined that the younger mice excreted Sr^{85} more rapidly than the older mice.

The retention of Sr^{85} in a castrated mouse depended on the time it was injected after castration. In the group injected with Sr^{85} two weeks after castration, there was no difference between this group and the control group. There was a definite increase in retention of Sr^{85} in the group injected four weeks after castration, and this was more marked in the group injected eight weeks after castration. The amount of Sr^{85} retained in the latter group is comparable to the calcium loss. The increased retention of Sr^{85} during the period of calcium loss is believed to be due to a hetero-ionic replacement of calcium ions by the strontium ions.—*Charles W. Cooley, M.D.*

MISCELLANEOUS

MAESTRI, A., MARCATO, M., NERI, F., and TAVERNA, L. Irradiazione cardiaca quale tentativo di prevenzione dell'infarto; studio arteriografico della circolazione coronarica in cani sottoposti ad infarto sperimentale previo trattamento radiante. (Irradiation of the heart as a possible preventative of infarction; arteriographic studies of the coronary circulation in dogs with experimental infarct which was preceded by radiation therapy.) *Ann. radiol. diag.*, 1960, *33*, 426–452. (From: Istituto di Clinica Chirurgica Generale e Terapia Chirurgica dell'Università di Parma and Istituto di Radiologia Medica dell'Università di Parma, Parma, Italy.)

The purpose of this experiment was to study the effects of moderate doses of radiation on the heart, followed by ligation of the descending branch of the anterior coronary artery to produce an acute coronary occlusion.

Thirty dogs were used in this experiment: 10 dogs received 1,500 r to the cardiac area; 10 received 2,000 r, and 10 were used as controls. Following irradiation of the 20 dogs, all, including the controls, were subjected to ligation of the descending branch of the anterior coronary artery. The ligature was placed at various sites, such as at the origin of the vessel, about 2 cm. distal to the origin, or at the greatest possible distance from its origin.

Recorded results concerned survival and the establishment of collateral circulation in the myocardium. Coronary angiography was used either *in vivo* or in autopsy specimens to study the vascular bed. The results show a higher percentage of survival in the irradiated group than in the controls. There was no appreciable difference between the dogs receiving 1,500 r or 2,000 r. Coronary arteriography showed that collateral and compensatory circulation developed to a greater degree in the irradiated myocardium.

The authors conclude that the myocardium which

has received a moderate dose of radiation can better cope with an acute coronary occlusion, because of its increased ability to establish collateral circulation, than the nonirradiated myocardium.

This is a very interesting and thought-provoking article. The illustrations are numerous and of excellent quality but perhaps could be more convincing. The authors are to be complimented for their excellent handling of the details of such a delicate experiment.—*Peter E. Russo, M.D.*

Maestri, A., and Dazzi, C. Ricerche sperimentali microradiografiche sulla vascolarizzazione del miocardio in condizioni di normalità e dopo trattamento radiante. (Experimental microradiographic research on the vascularity of the myocardium of the normal heart and following radiation therapy.) *Ann. radiol. diag.*, 1960, *33*, 453–470. (From: Istituto di Radiologia dell'Università di Parma, Parma, Italy.)

The authors state that previous experiments concerning the heart and coronary circulation showed that moderate doses of radiation produce hyperemia and favorably improve the vascularity of the organ. The results were evaluated arteriographically. In the present experiment, microangiographic methods were employed to study these changes in the vascular supply of the myocardium.

A group of 20 dogs was used. A dose of 2,000 r, with conventional treatment factors, was given over the cardiac area. In 10 dogs the dose was protected over a period of twenty days; the other 10 dogs were used as controls. About a week after irradiation was completed, all of the dogs were sacrificed.

A solution of barium sulfate was used to inject the coronary vessels of the heart. The pressure applied to this injection was sufficient to fill the most terminal capillaries. In none of the specimens was contrast medium found in the venous side of the circulation. After the injection the coronary arteries were ligated at their origin. In some of the specimens, prior to the injection of the opaque material, the descending branch of the anterior coronary artery was ligated in an effort to demonstrate, if possible, the collateral circulation to this occluded vessel. The injected hearts were kept at temperatures below 0° to solidify the contrast medium and to prevent compression of the smaller vessels during the preparation of the microscopic sections. The preparations were then fixed in 10 per cent formalin. Microradiographs of these specimens were studied for vascularity of the myocardium.

The authors arrived at the following conclusions: Irradiation of the myocardium with a certain dose produces vasodilatation of the coronary arteries, especially at the precapillary level. Also the collateral and communicating branches become more functional. These terminal collateral and communicating branches vary from 20 to 200 μ in width and 2 to 3 cm. in length; they can also be demonstrated in the myocardia which did not receive irradiation but are smaller in caliber and less numerous in number. Following ligation of the descending branch of the anterior coronary artery, anastomosis with adjacent vessels in the affected area can be shown. These collateral and communicating branches are not newly formed vessels but are present in a latent state.

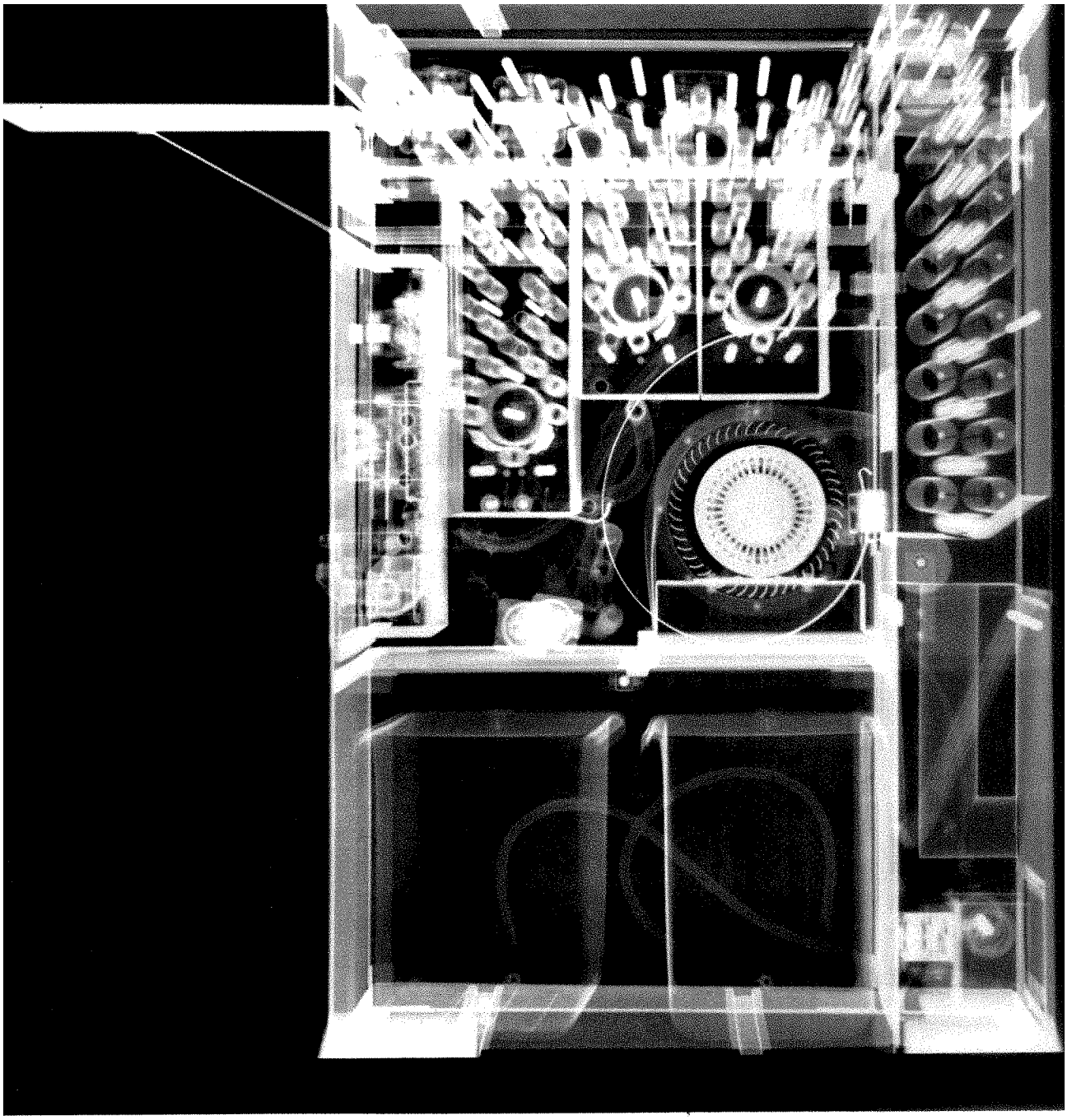
The article contains many illustrations of microradiographs which appear to be rather convincing. It is, however, disturbing to know that irradiation, in moderate doses, is used to control vascularization in tissues such as the cornea and conjunctiva.—*Peter E. Russo, M.D.*

Levy, Marie L., Palazzi, Hugo M., Nardi, George L., and Bunker, John P. Hepatic blood flow variations during surgical anesthesia in man measured by radioactive colloid. *Surg., Gynec. & Obst.*, Mar., 1961, *112*, 289–294. (From: The Anesthesia Laboratory and the Department of Surgery of the Harvard Medical School at the Massachusetts General Hospital, Boston, Mass.)

Hepatic blood flow was studied in 68 patients undergoing a variety of surgical procedures during thiopental, cyclopropane, ether, local and spinal anesthesia.

Hepatic plasma flow was determined by measuring the clearance of injected radioactive colloid chromic phosphate (P^{32}) by the reticuloendothelial system of the liver.

The following conclusions were reached: premedication with pentobarbital and atropine produces only slight changes. There is depression in liver blood flow during general and spinal anesthesia. Changes in liver blood flow reflect changes in arterial pressure, in portal venous pressure and in vascular resistance within the liver. During thiopental and cyclopropane anesthesia, changes in hepatic plasma flow were consistent and significant. During ether anesthesia, changes were variable and of small magnitude. This is explained by the release of epinephrine and norepinephrine into the blood with ether as an anesthetic agent. At the end of anesthesia, the difference between various agents disappears and the final mean hepatic blood flow is very nearly the same for all agents. Cirrhotic and noncirrhotic livers respond similarly.—*Bernard Loitman, M.D.*



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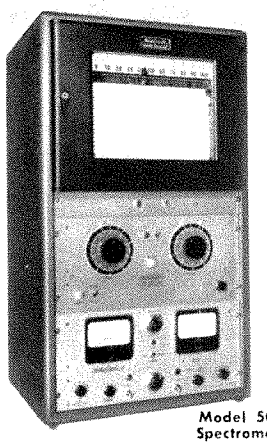
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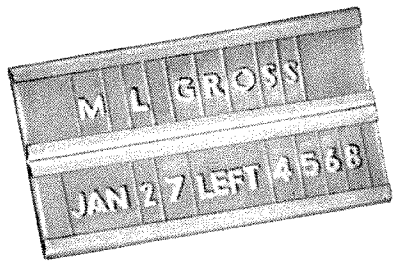
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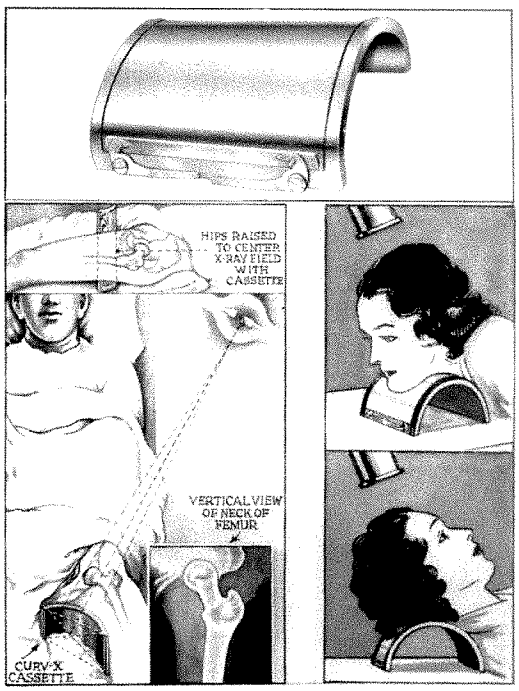
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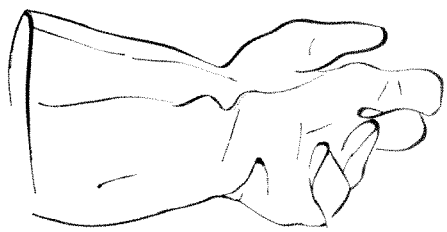
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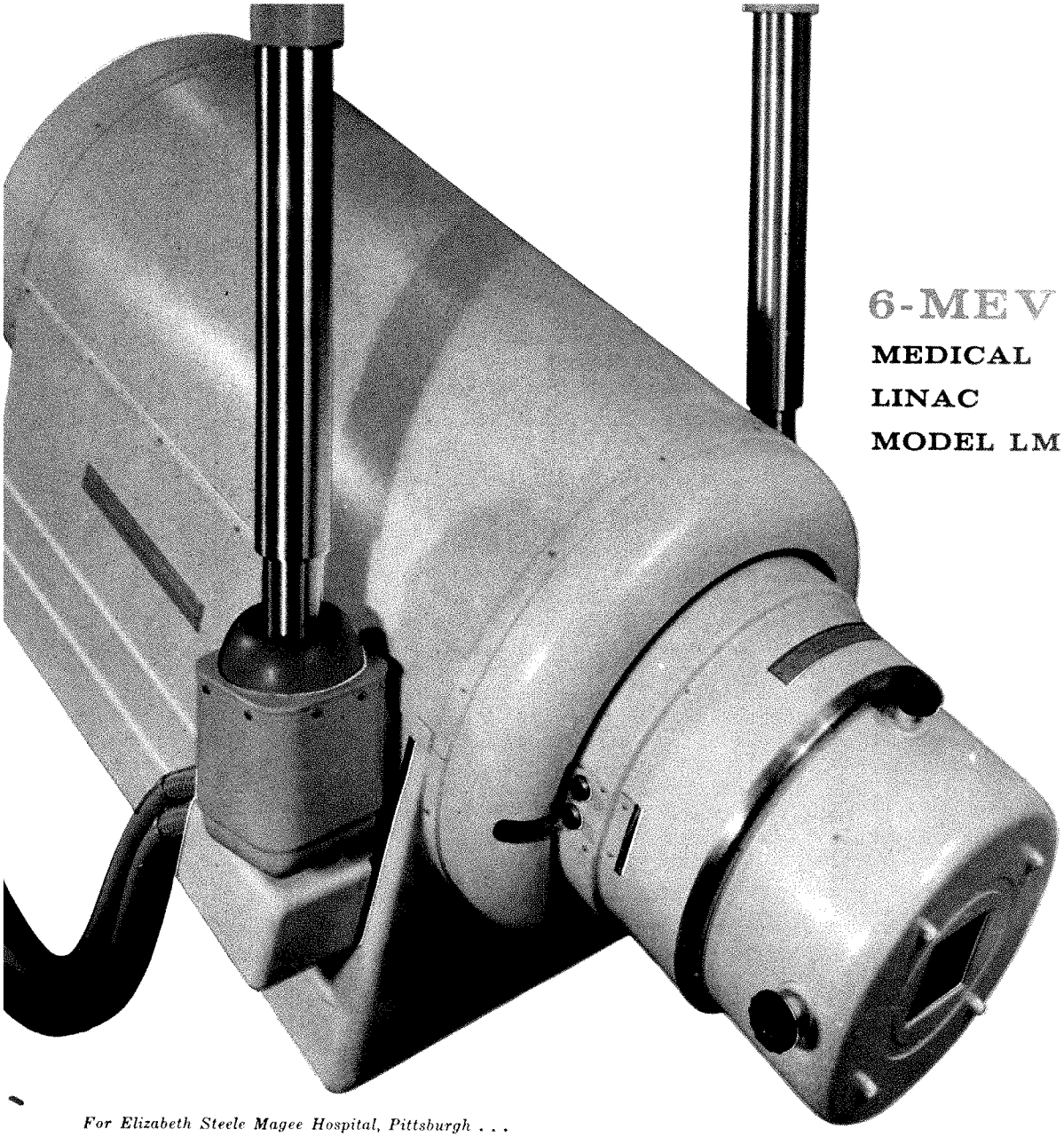
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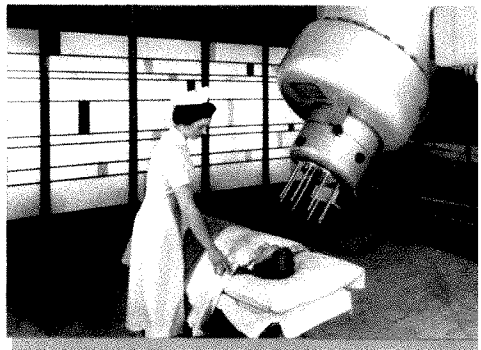


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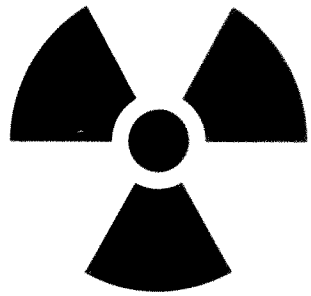
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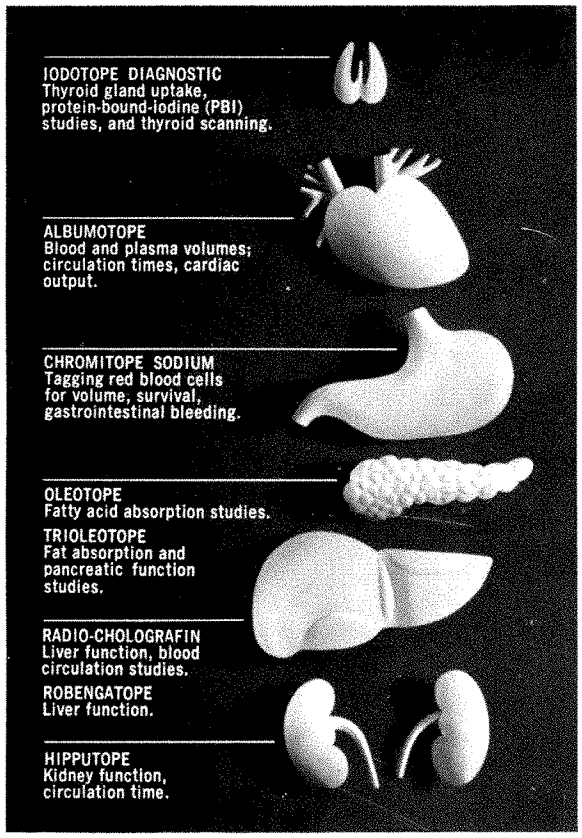
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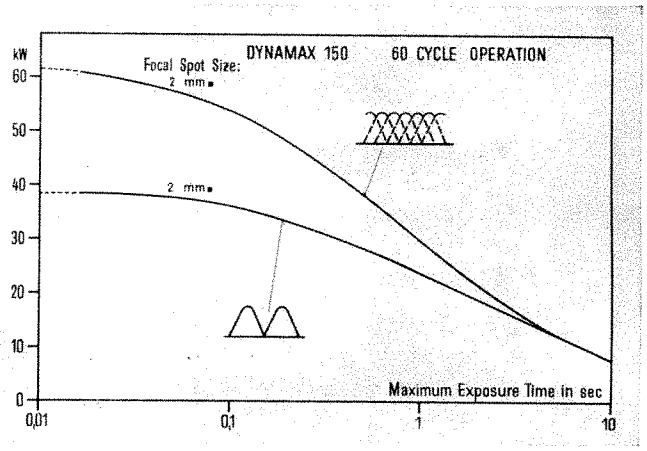
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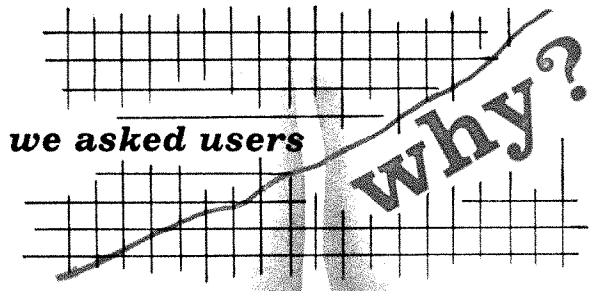
2. the x-ray tube is more effectively utilized; e.g. a Dynamax 150, at 0.1 sec, has a maximum permissible load of 54 and 36 kW for three and single-phase operation respectively (see curves).



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ap	15 cm	90	12,5	0.02	85	8	0.006
lat	18 cm	100	12,5	0.024	95	8	0.007
Adult							
ap	23 cm	100	12,5	0.024	95	8	0.012
lat	32 cm	115	12,5	0.027	110	8	0.013

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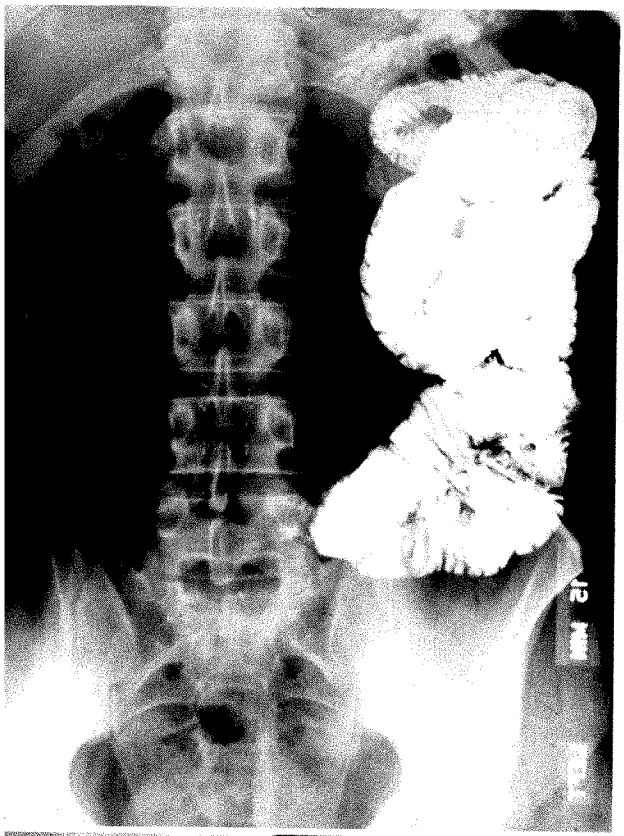
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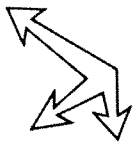
*BAROTRAST



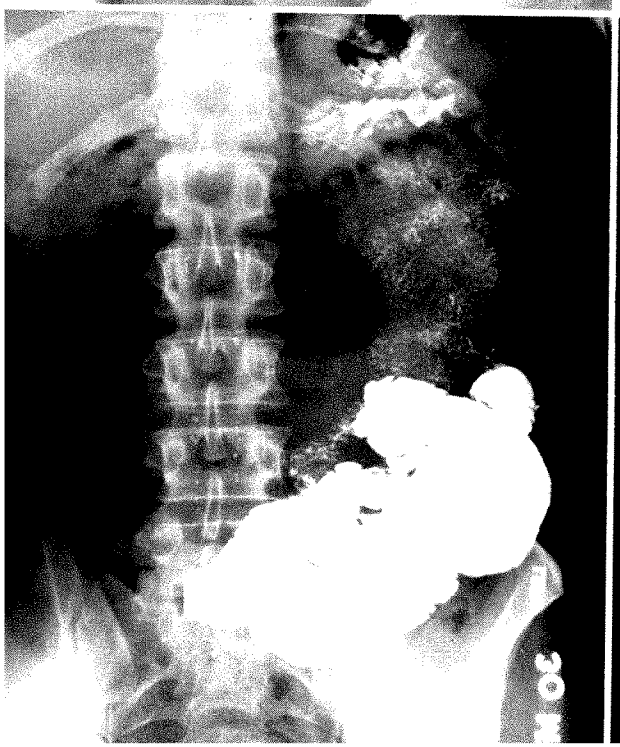
BAROTRAST will flow through the small bowel as a column without clumping, precipitating, or forming concretions.

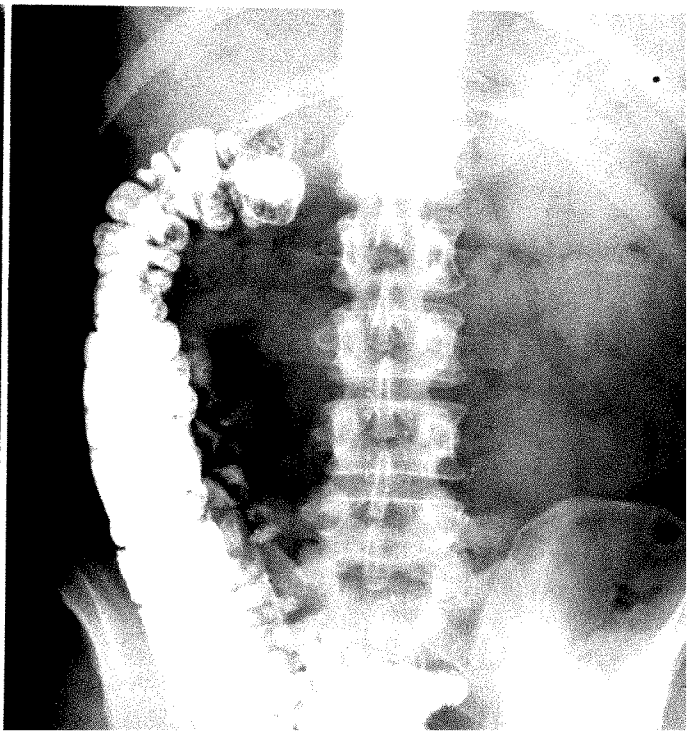
BAROTRAST, without any additive or chilling, will normally pass through the small bowel and into the colon in 2 to 3 hours, about half the usual time required when using barium sulfate, U.S.P.

BAROTRAST flow properties are not altered by intestinal environmental change.



Progress X-rays show excellent delineation and "columnar" movement of BAROTRAST through the small bowel.





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X-RAY NEWS

No. 52

Special view of the zygomatic arches and facial bones

by John G. Gines, R. T. (A.R.X.T.), St. Luke's Hospital, San Francisco, California

In radiography of the Zygoma these views are routine: (1) Basal view (with a lighter exposure to demonstrate the facial bones better); (2) modified AP Towne; (3) Waters; (4) oblique.

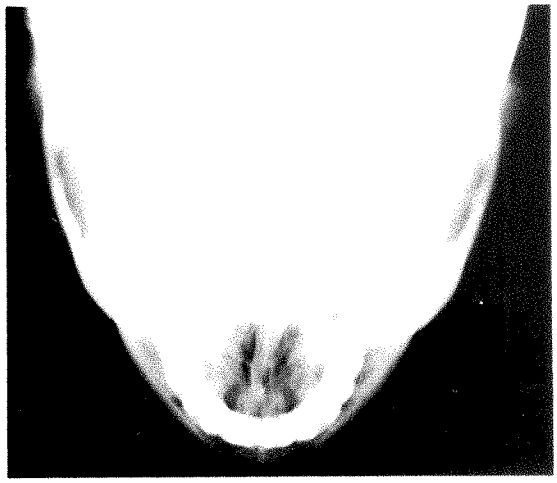
In some cases these routine views fail to demonstrate disease or fracture adequately. The following technique was developed for just such cases.

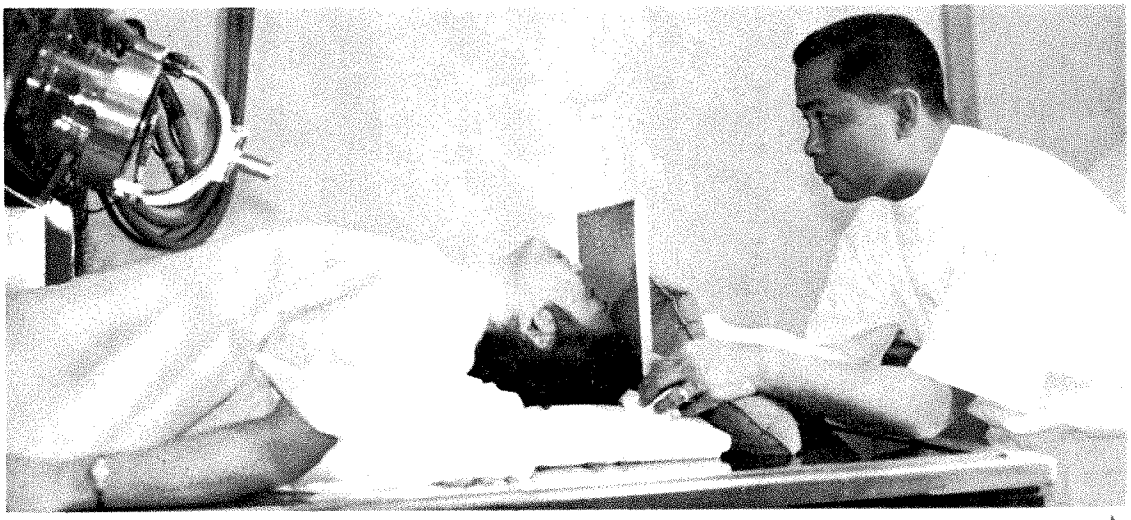
With the patient supine on the radiographic table (see photo at bottom), the shoulders are raised on a folded pillow. The head is allowed to fall back until the vertex of the skull is in firm contact with a soft pad. This gives you a position similar to a submento-vertex position.

The tube angle will vary with the degree of extension of the individual patient's neck. The cassette is placed crosswise against the vertex of the skull, perpendicular to the table top, with the lower edge of the cassette resting on a pad. A sandbag supports it from behind.

The central ray is centered midway between the zygomatic arches. It should pass along a line connecting the angle of the mandible with the outer canthus of the eye. The exposure is 100 MA—1/10 second—66KvP, at 36-inch distance, where the head measures 24 cm from vertex to angle of mandible.

This view will demonstrate these structures unusually well:





OVER

1. Zygomatic arches
2. Hard palate and greater palatine foramina
3. Anterior walls of the maxillary sinuses
4. Anterior body of the mandible and symphysis

Tips for Technicians

Care of processing hangers

by **Neil J. Lyons, R. T. (A.R.X.T.)**
Drs. Woolley, Burton, Hunter & Lee
Portland, Oregon

In order to do any job correctly with the least amount of effort, it is essential to have the necessary equipment in good working order. The processing of x-ray film is not only hindered by hangers in poor condition, but the quality of the radiograph often suffers, because tiny bits of gelatine or film adhering to the clips will absorb chemicals and cause streaks on the film during development. There are two things which must be done periodically if the hangers are to be kept in good repair. They must be adjusted and they must be cleaned.

The least expensive and most effective method for removing the accumulated emulsion is to soak the hangers in a solution of Spic and Span, a common domestic cleansing powder. Eight to ten tablespoons of the powder are dissolved in five gallons of warm water. After the hangers have remained in this solution overnight, the pieces of old film adhering to the clips can be removed with a knife. A small piece of cloth is then wrapped around the blade of the knife and used to wipe off the inner surface of the clip. Thorough rinsing of the hangers is required before returning them to operation.

As each clip on a hanger is cleaned, it should be checked for poor alignment and malfunction due to a lack of tension in the spring portion. Any clip which has not had the pin broken off can be restored to normal function with little effort.

A pair of long-nosed pliers similar to those used by electricians is required for realignment. The first step is to restore the tension to the spring. This is accomplished by grasping the curved end of the clip with the pliers and applying enough pressure to increase the curve on the end and widen the space between the sides of the clip. The pliers should be inserted from below when adjusting the upper clips and from above in adjusting the lowers. This maneuver will restore the tension to the spring and also realign the part of the clip which holds the film. In many instances, this is all that is required. However, if the two flat surfaces are not in good contact, a little more manipulation is needed. In order to get the pin on one side of the clip to project through the center of the hole on the other, it is sometimes necessary to flatten out the two parts with the pliers. Occasionally a change in the angle on the arms leading to the main portion of the clip is also required. With a little practice, it is easy to determine where to straighten a clip and how much pressure is needed.

Practically all of the hangers used today possess the same basic clip design. On some types, it is a little more difficult to insert the tip of the pliers into the area to be straightened, but all makes can be returned to service after realigning the clip and increasing the tension on the spring.



"NEXT!!"

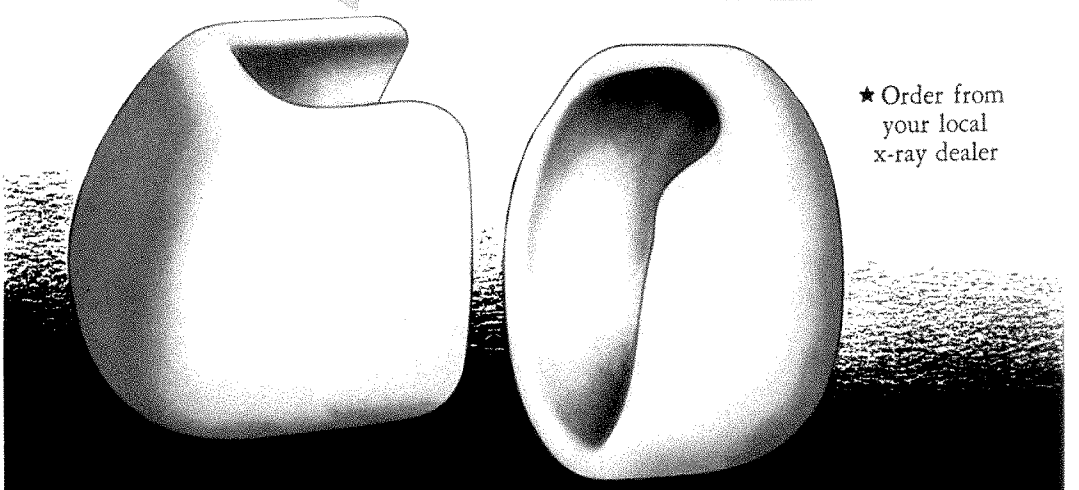
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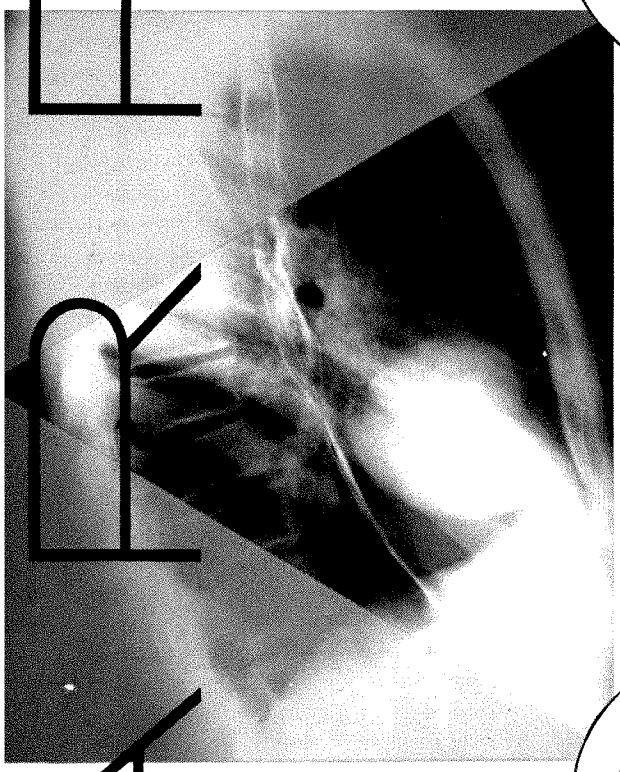
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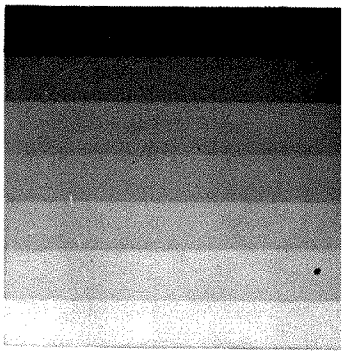
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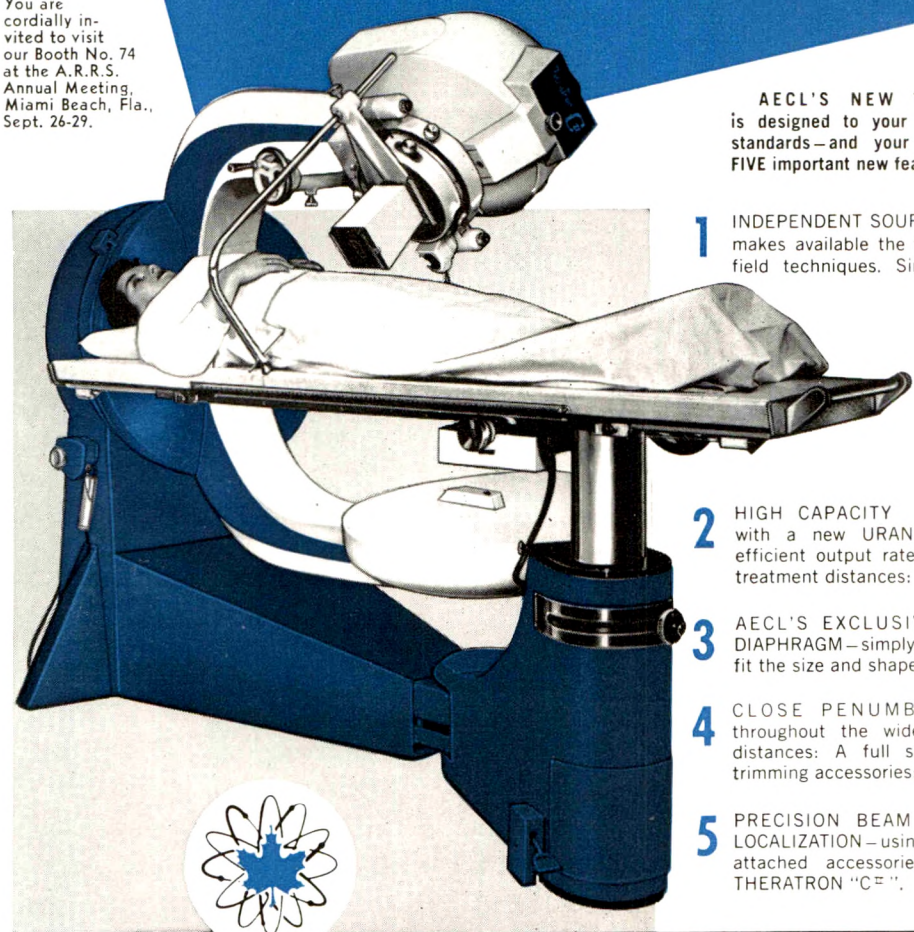
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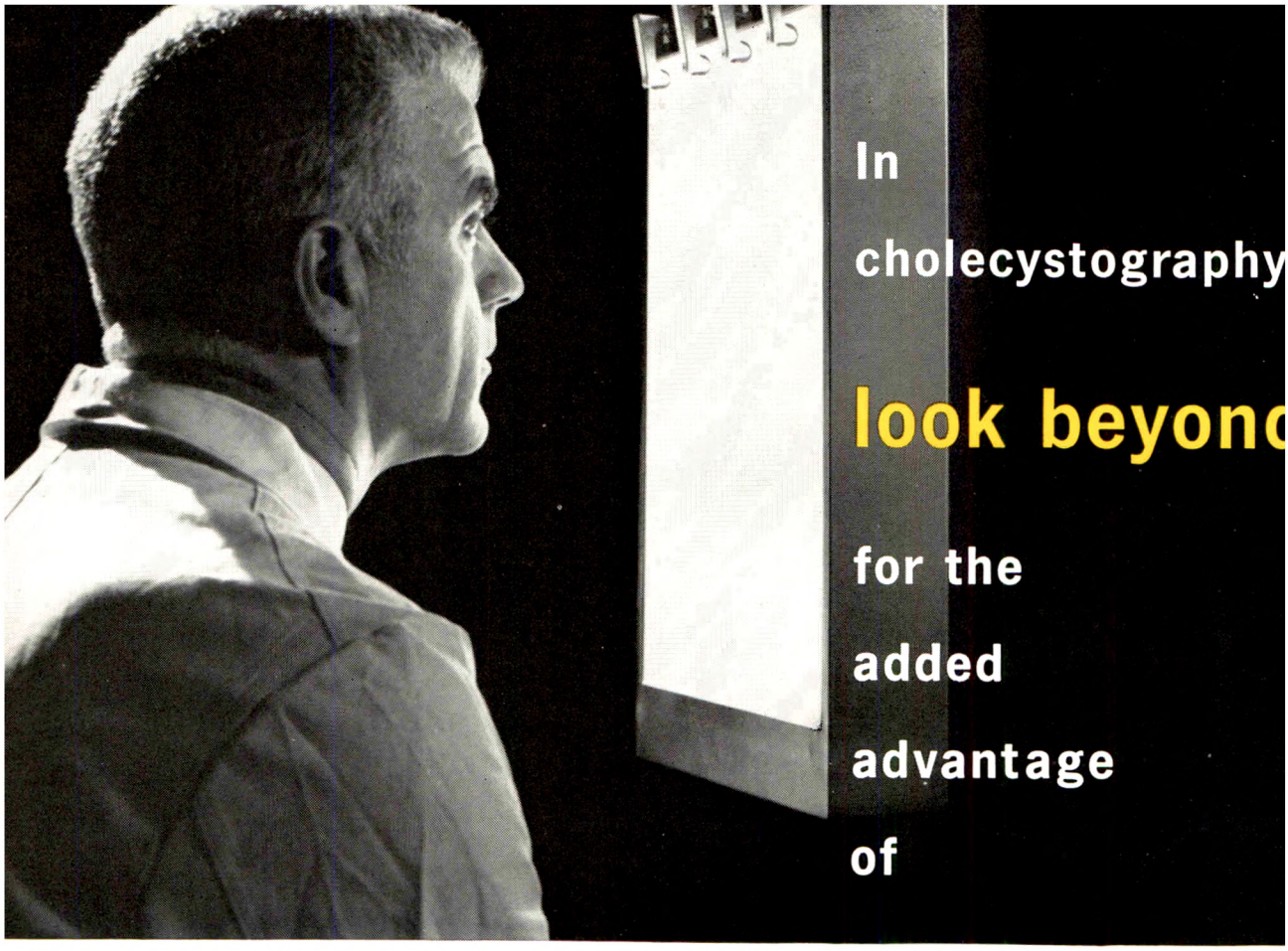
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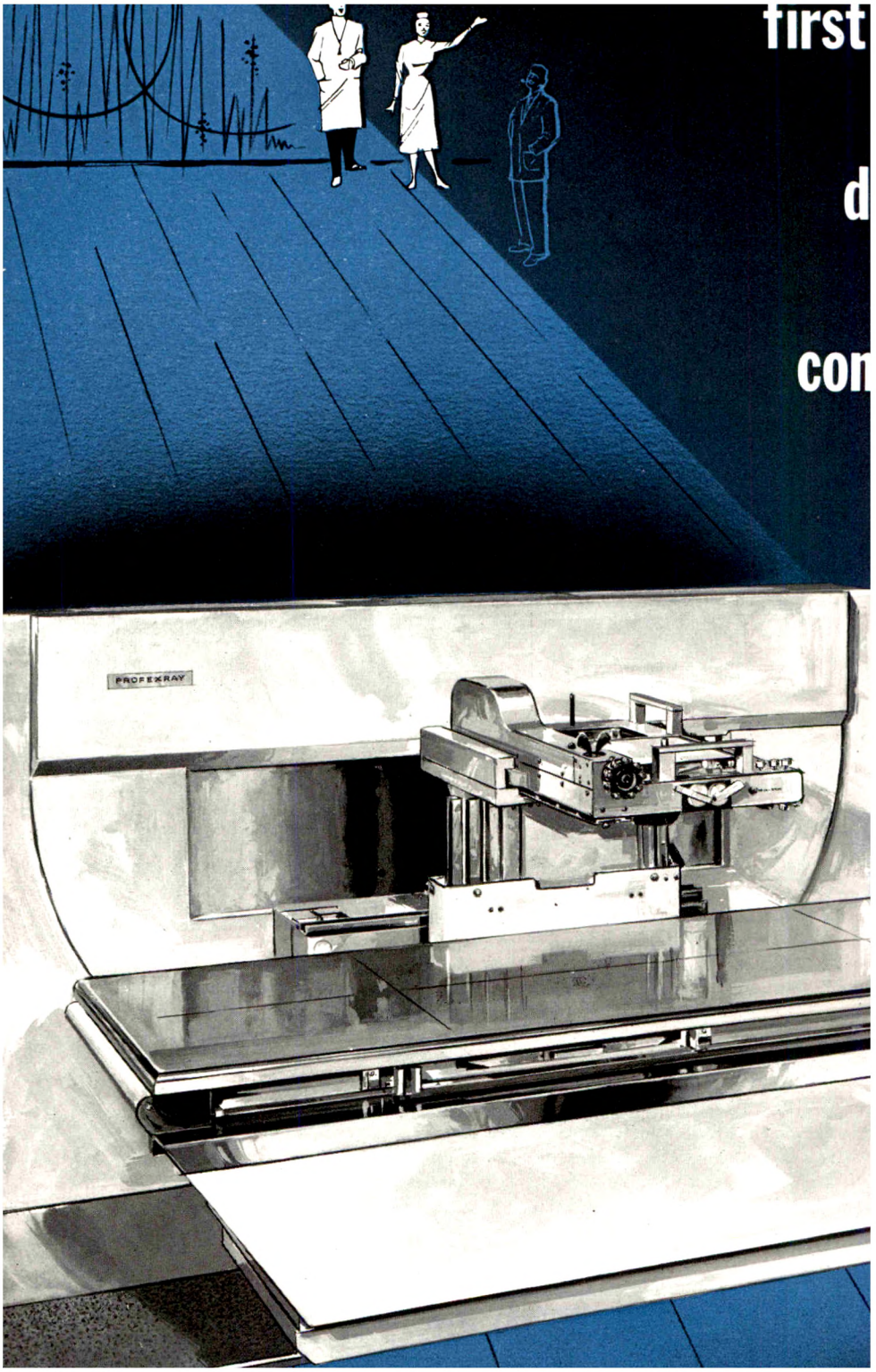

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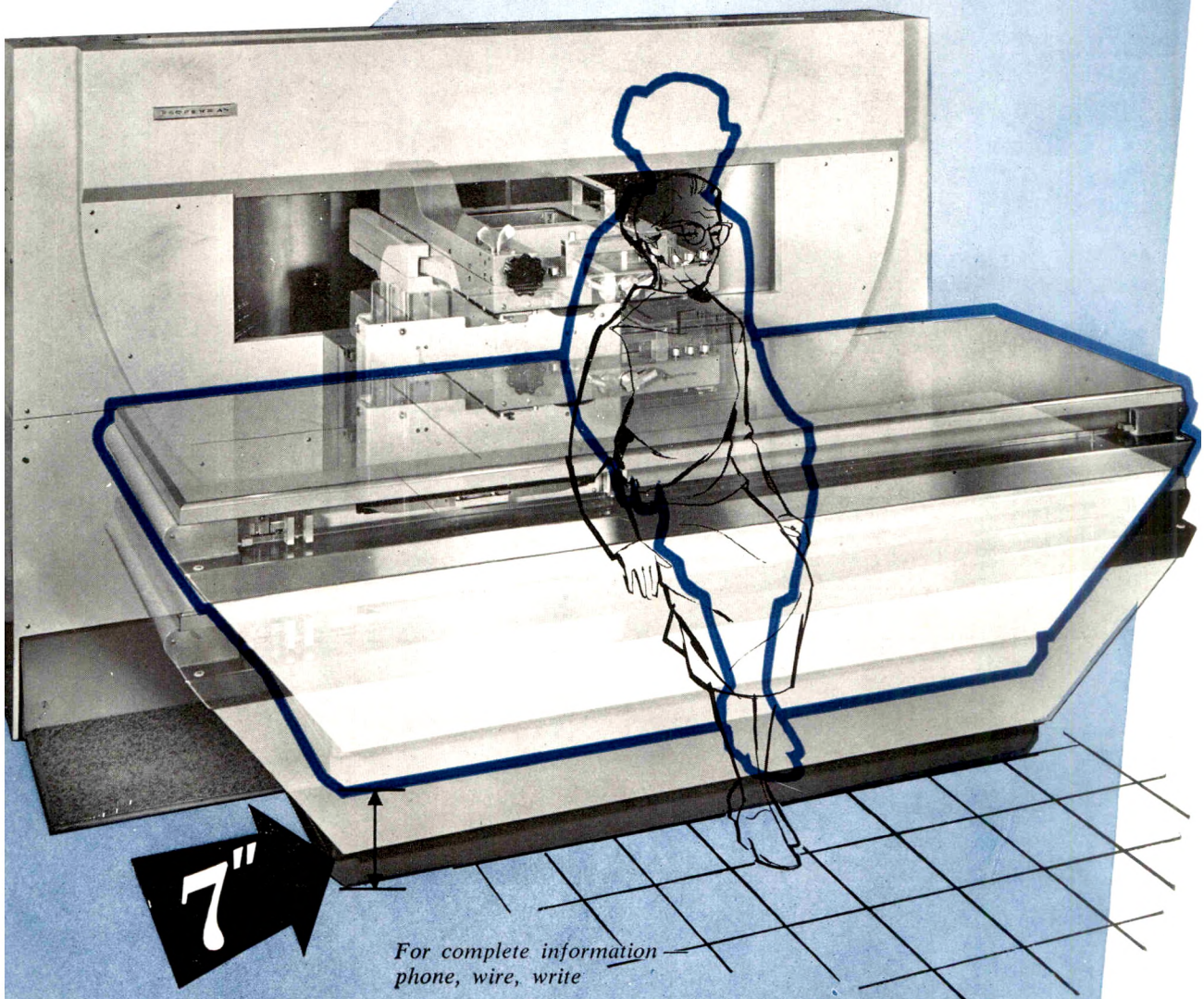
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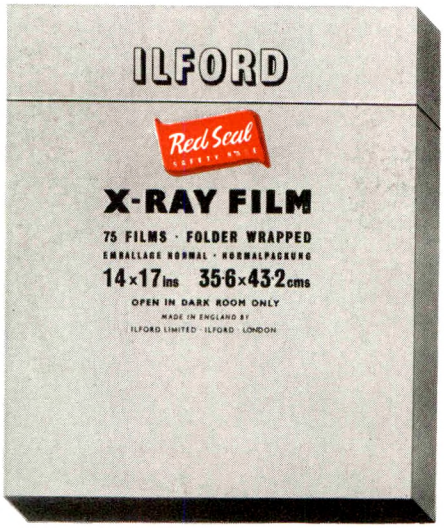
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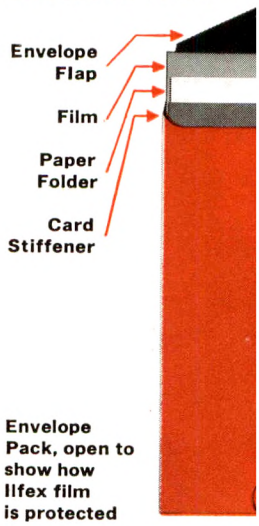


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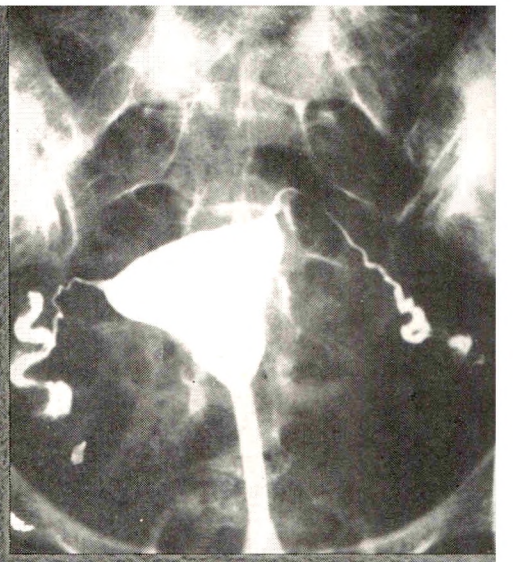
This ultra-fast high-spe
vides bone and tissue det
that which can be obtai
tional screen techniques
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other non-screen film!
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graphs. Boxes of 25, in

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NEW MINIROL-X PROCESSOR

SMALL 44 inch long base

EFFICIENT Fast 7 minute uniform processing

LOW COST Priced to fit any X-Ray department

The Minirol-X is a proven, automatic system for processing and drying X-Ray film the hangerless way—complete roller operation. Occupies less than 10 sq. ft. of space. It is designed for efficient, economical processing in the private office or clinic, and small as well as large hospitals who wish to diversify processing stations. Let the Pako Planning Service show how a Minirol-X Processor will fit your X-Ray department. For complete information contact your Pako Distributor or write to Pako Corporation.



Minirol-X Processor—Left Hand Installation Illustrated

THE TINY MINIROL-X HAS BIG FEATURES:

- Full worm driven racks, no chains
- Right and left hand installation
- Quiet, roller-type operation
- 7-minute total processing time
- No. 4 Satin Stainless Steel finish
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- Processes all brands of medical screen film in sizes from 4" x 4" thru 17" x 36" sheet film. Roll film in widths of 17" or less.

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(fully compatible with automatic processing techniques)

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That is why today more and more leading radiologists are specifying Ansco High-Speed X-ray Film. Ansco, Binghamton, N. Y. A Division of General Aniline & Film Corporation.

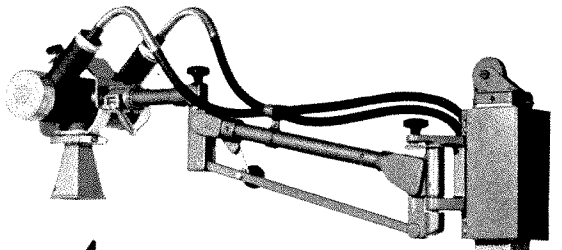
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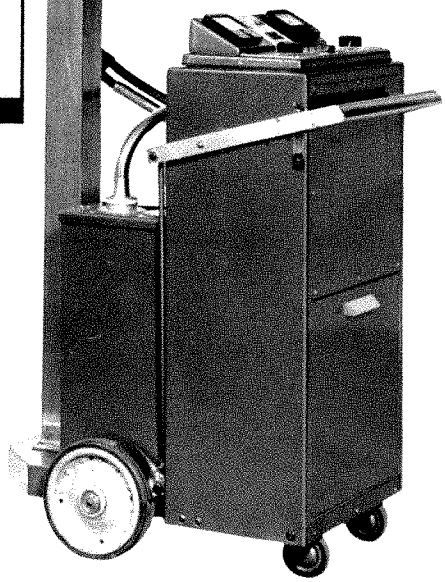
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MODEL GR-200 MOBILE X-RAY UNIT



TUBE EXTENDS FULL 48 INCHES FROM TUBE COLUMN

- capacity: 200 Ma at 100 P.K.V.
- full wave rectified . . . 4 valve tubes



Large 10 inch diameter, rubber tired roller bearing wheels make unit so easy to move and maneuver.

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Unit operates on 220 or 110 volt line.

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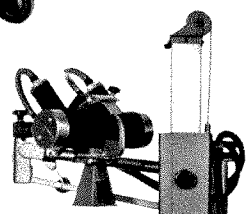
- Unit also available in 100 Ma at 100 P.K.V. Full Wave Rectified Capacity.

Standard

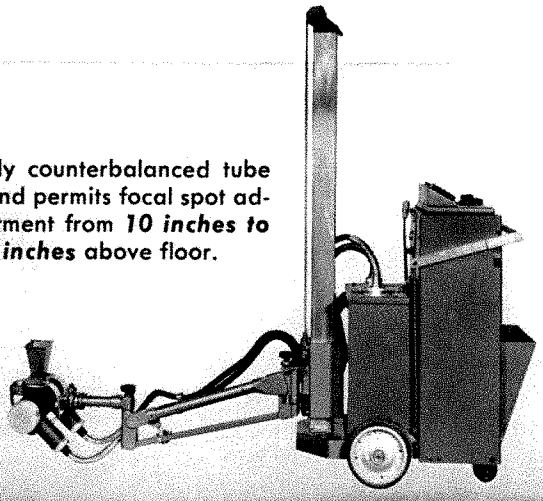
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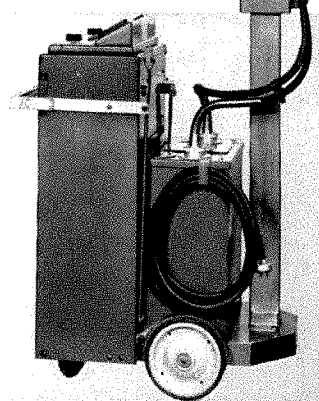
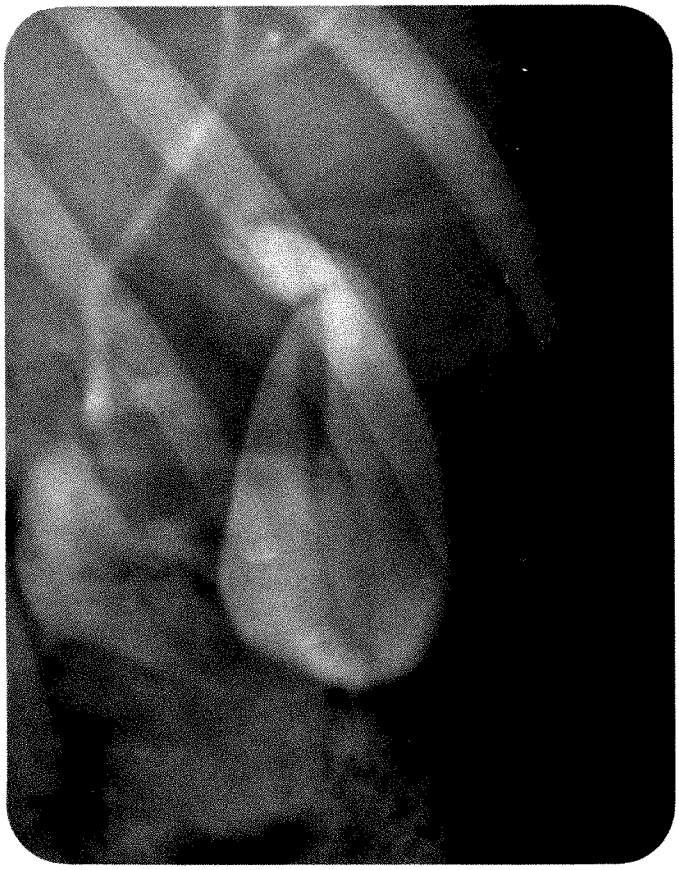


Illustration shows tube "parked" for easy moving or storage.

"Outstanding Contribution"[1] in Cholangiography



Rapid Optimal Opacification: Within 15 minutes, Cholografin outlines hepatic and common ducts even after cholecystectomy, reveals biliary ducts in about 25 minutes, and completely opacifies the gallbladder within 2½ hours.[1-4]

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Duografin Squibb Diatrizoate and Iodipamide Methylglucamines for rapid visualization of biliary *and* renal tracts in routine examinations or differential diagnosis

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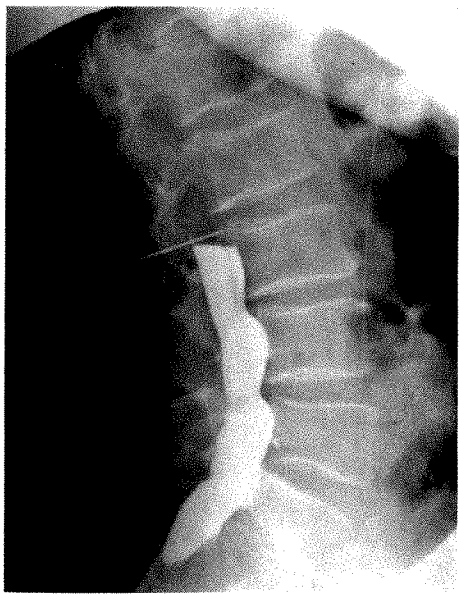
Duografin is supplied in bottles of 50 cc.

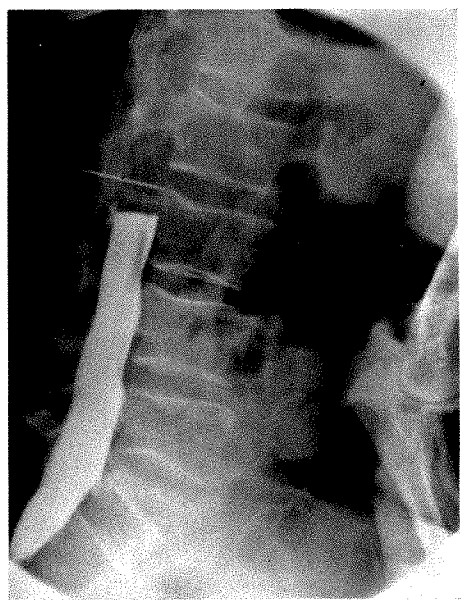
For full information, see your Squibb Product Reference or Product Brief.

References: (1) Cohn, E. M.: Am. J. Gastroenterol. *35*:115 (Feb.) 1961. (2) Jones, M. D.; Sakai, H.; and Rogerson, A. G.: J. Pediat. *53*:172 (Aug.) 1958. (3) Machella, T. E.: Gastroenterology *34*:1050 (June) 1958. (4) Orloff, T. L.: Am. J. Roentgenol. *80*:618 (Oct.) 1958. (5) Johnson, G., Jr.; Pearce, C.; and Glenn, F.: Ann. Surg. *152*:91 (July) 1960. (6) McClenahan, J. L.: Pennsylvania M. J. *62*:188 (Feb.) 1959.

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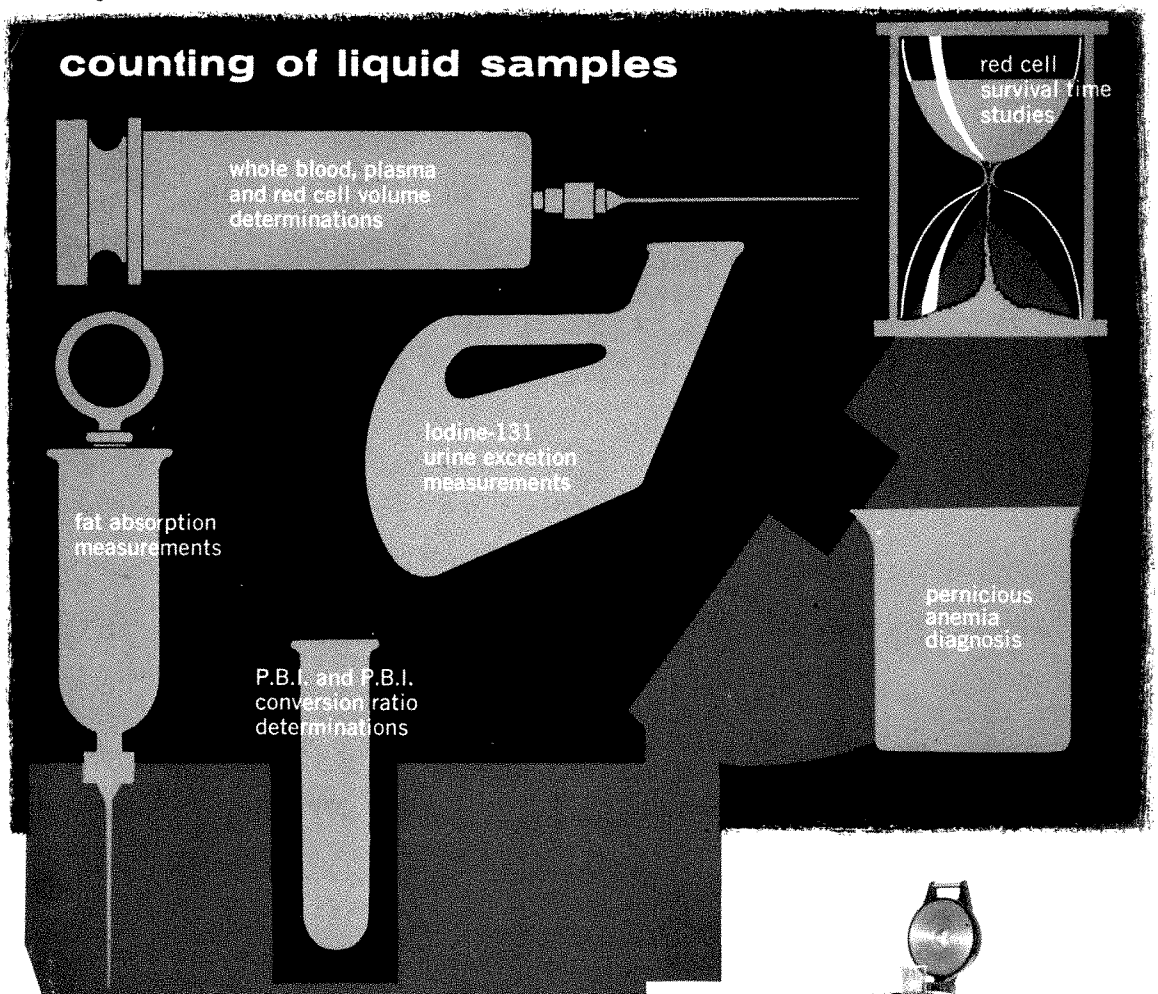


Dynamic Myelography

These two radiographs of the same patient demonstrate the bulging of the annulus fibrosus during hyperextension and flexion, respectively, of the vertebral column. 30 cc of ***Pantopaque*** contrast medium was used. Note how this technique permits visualization of the posterior surface of the vertebral canal.

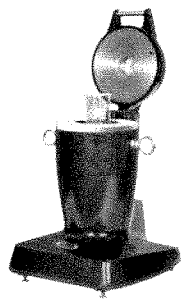
"PANTOPAQUE" *is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized by the Research Laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iophendylate Injection, U.S.P., by Lafayette Pharmacal Inc. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*

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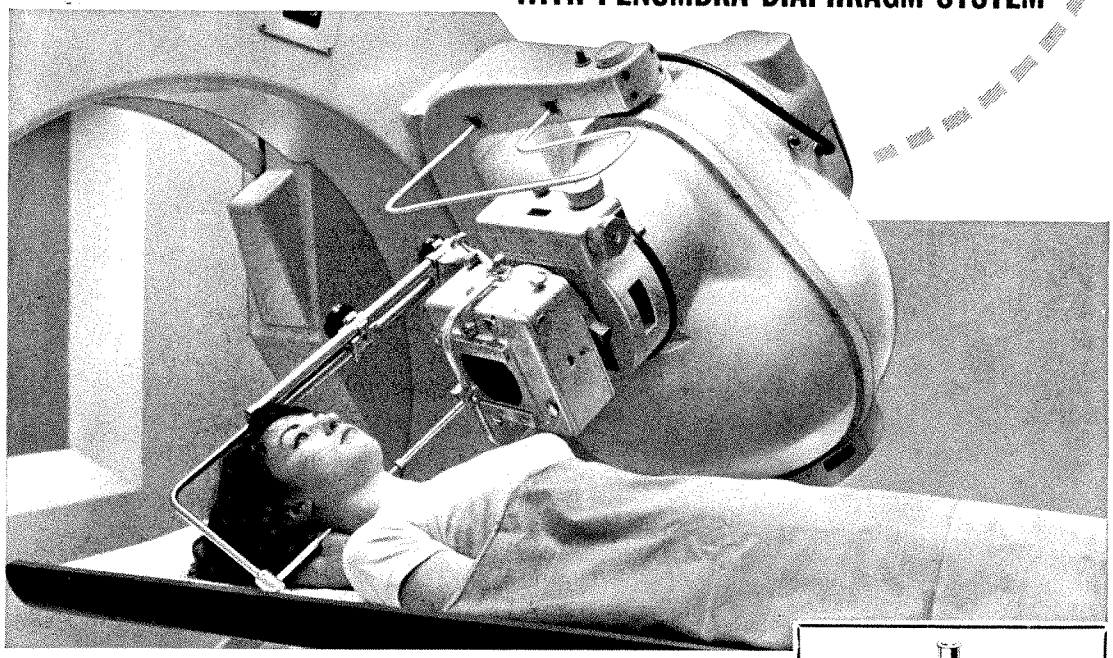
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'ORBITRON'

COBALT THERAPY EQUIPMENT

WITH PENUMBRA DIAPHRAGM SYSTEM

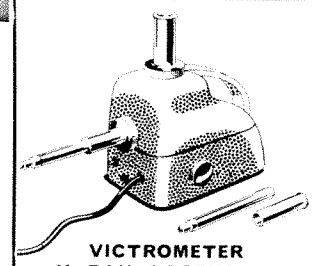


The penumbra diaphragm of the 'Orbitron' cobalt therapy equipment sharply defines the fields with diaphragm leaves at minimum distance from the skin. Easily attached, and with micrometer adjustment of tungsten copper blocks moving parallel to the sides of the beam, the penumbra diaphragm provides a far higher standard of beam definition than hitherto possible in cobalt therapy.

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A portable quartz fibre electrometer-type instrument for consistently accurate determination of X-ray doseage or intensity in r per min. Mains energised and entirely self-contained.

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Roller Processors

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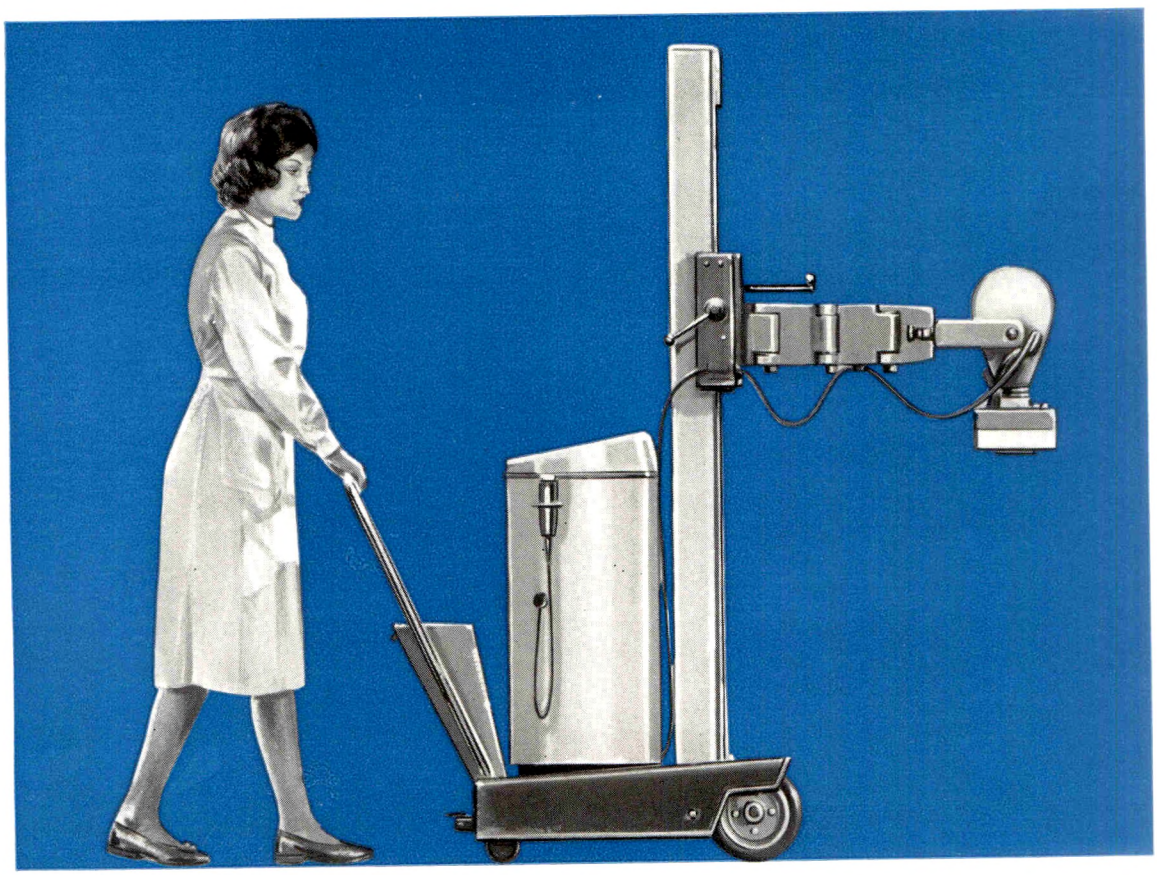
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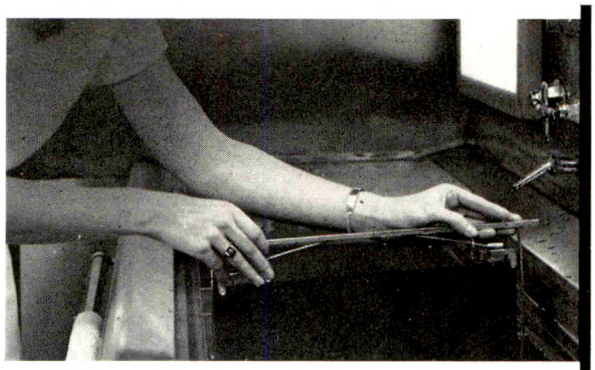
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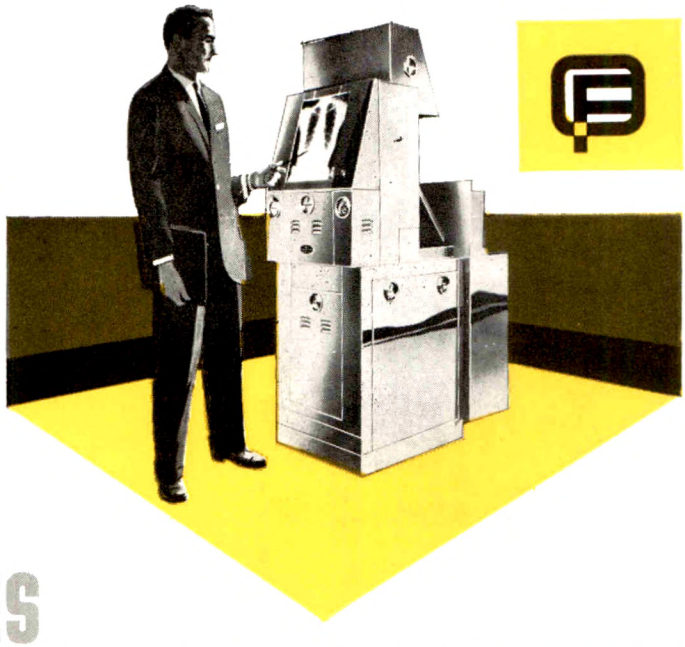
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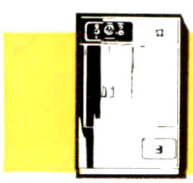
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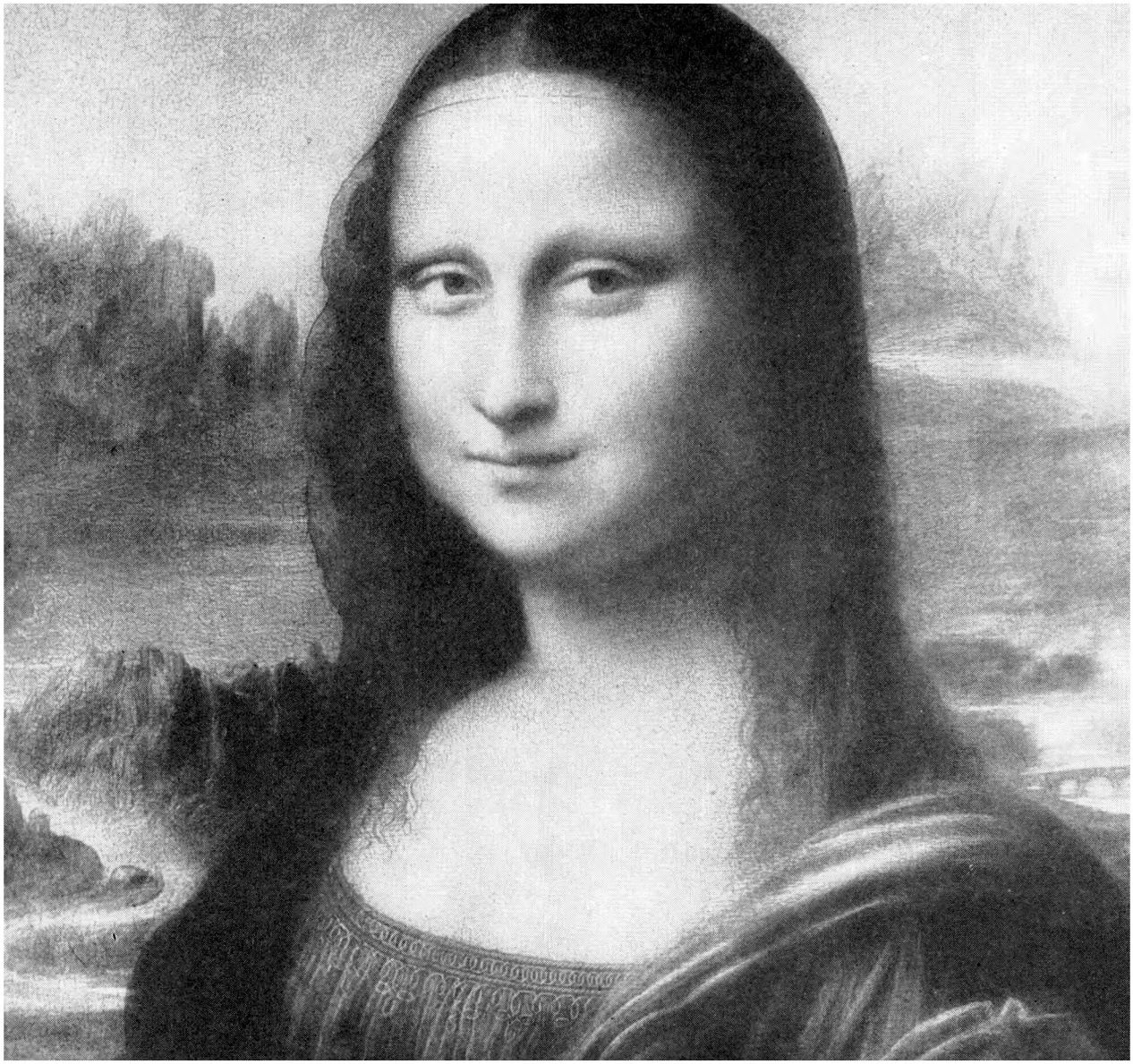
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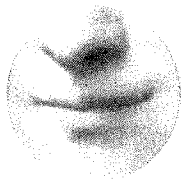


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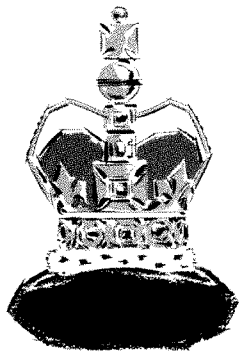
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Also Available: Hypaque-M, 75 and 90 per cent (concentrated solutions of sodium and methylglucamine diatrizoate) for angiocardiography, angiography (selected cases), nephrotomography and hysterosalpingography, vials of 20 and 50 ml.

References: 1. Lloyd, F. A., and Cottrell, T. L. (Northwestern Univ.): Diatrizoate sodium, *Ann. New York Acad. Sc.* **78**:987, July 2, 1959. 2. Lance, E. M., and Killen, D. A. (Vanderbilt Univ.): Experimental appraisal of the agents employed as angiocardiographic and aortographic contrast media. I. Neurotoxicity, *Surgery* **46**:1107, Dec., 1959. 3. Pool, J. L. (Columbia Univ.): Early treatment of ruptured intracranial aneurysms of the circle of Willis with special clip technique, *Bull. New York Acad. Med.* **35**:357, June, 1959. 4. Davis, D. R. (St. Luke's Hosp., Kansas City, Mo.): Operative cholangiography by utilizing the cystic duct, *Surg. Gynec. & Obst.* **112**:378, March, 1961. 5. Youngstrom, Karl A. (Univ. of Kansas): Study of use of diatrizoate sodium (Hypaque), *J. Kansas M. Soc.* **59**:308, July, 1958.

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. . . even during difficult procedures. An Imperial II diagnostic x-ray installation is oriented to the radiologist. This stop-motion color photo provides a graphic example: with ring-mounted pivoting, screen-to-eye relationship remains practically constant and centered . . . spot-film device neither advances nor recedes as table is tilted.

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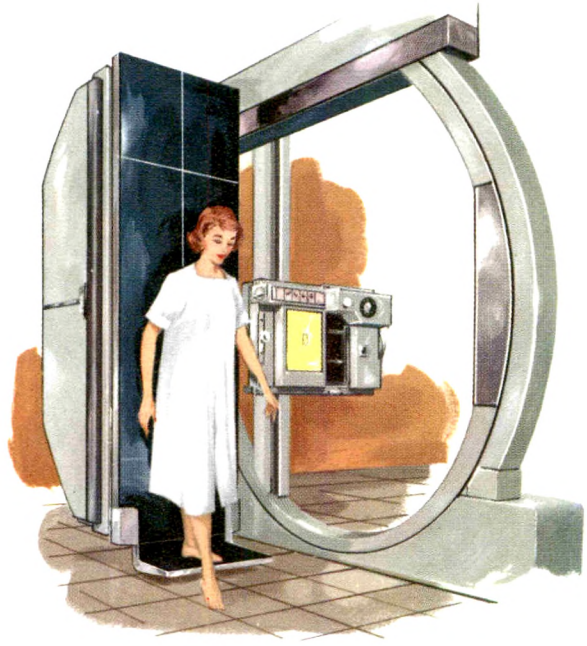
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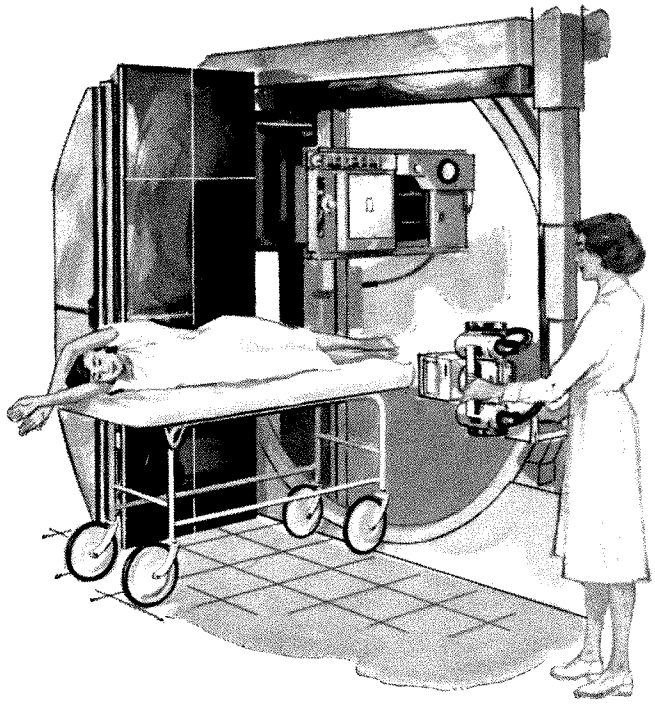


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. . . permits you to move patient and table laterally — without hands, without effort — without disturbing the position of the screen. Superbly balanced lateral shift is the reason for this handling ease . . . another almost-automatic way Imperial responds to your every touch.

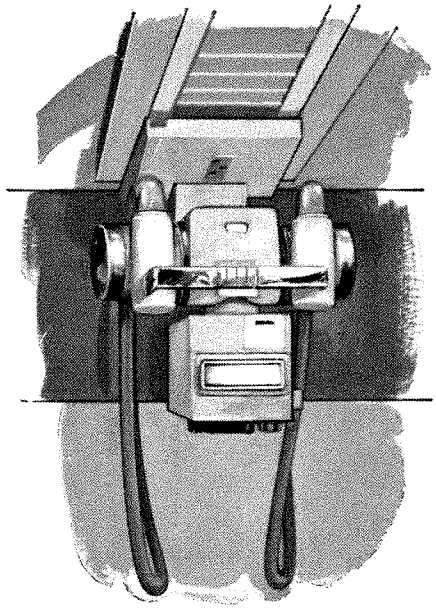


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. . . are reflected in outstanding flexibility for radiographic procedures in close quarters — a 12-ft. room, for instance. Tilting table — cradled in its unique double-ring mounting — actually retreats in the upright position. You can do radiography at preferred target-film distances (for example, 6-ft. chest procedures) when the table is vertical in either direction. Cart work, too, is easier with the table out of the way.



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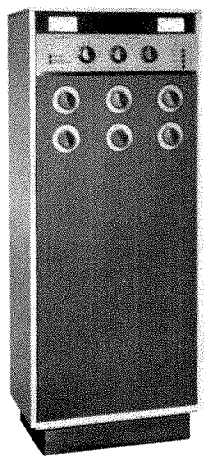
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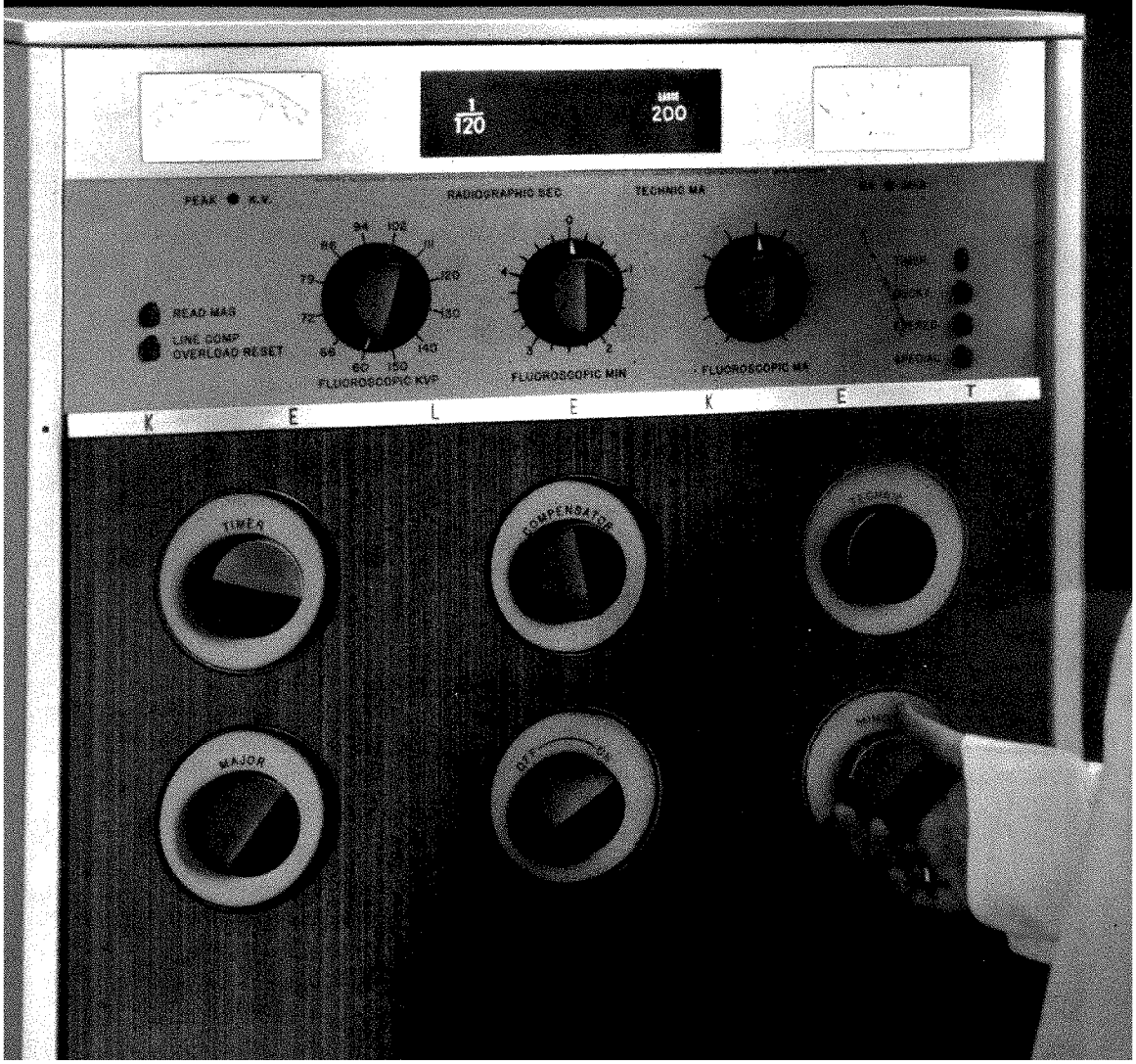
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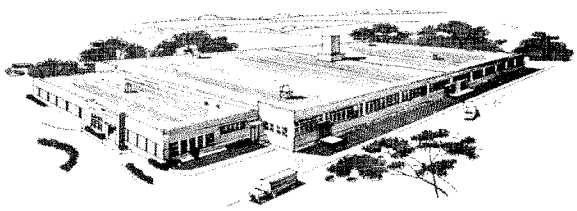
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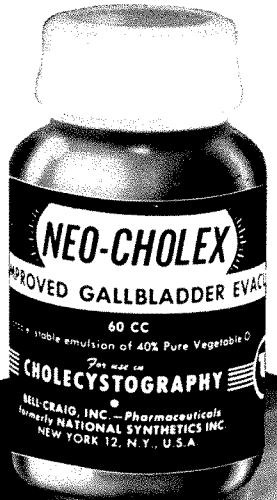
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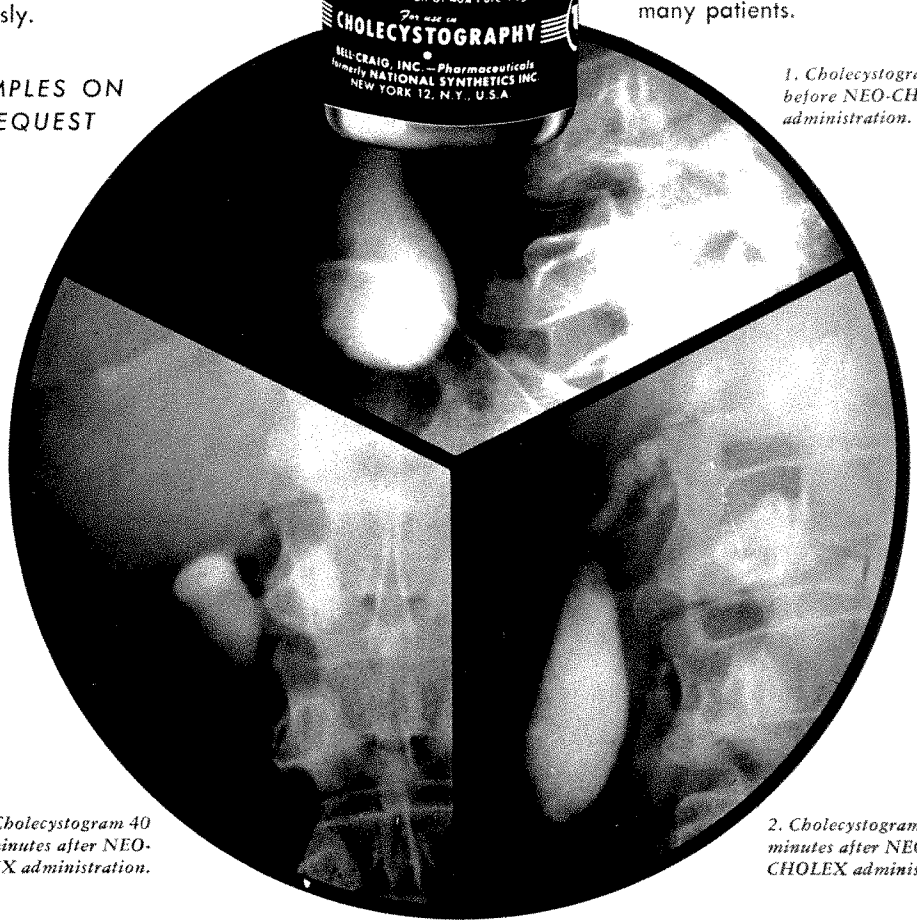
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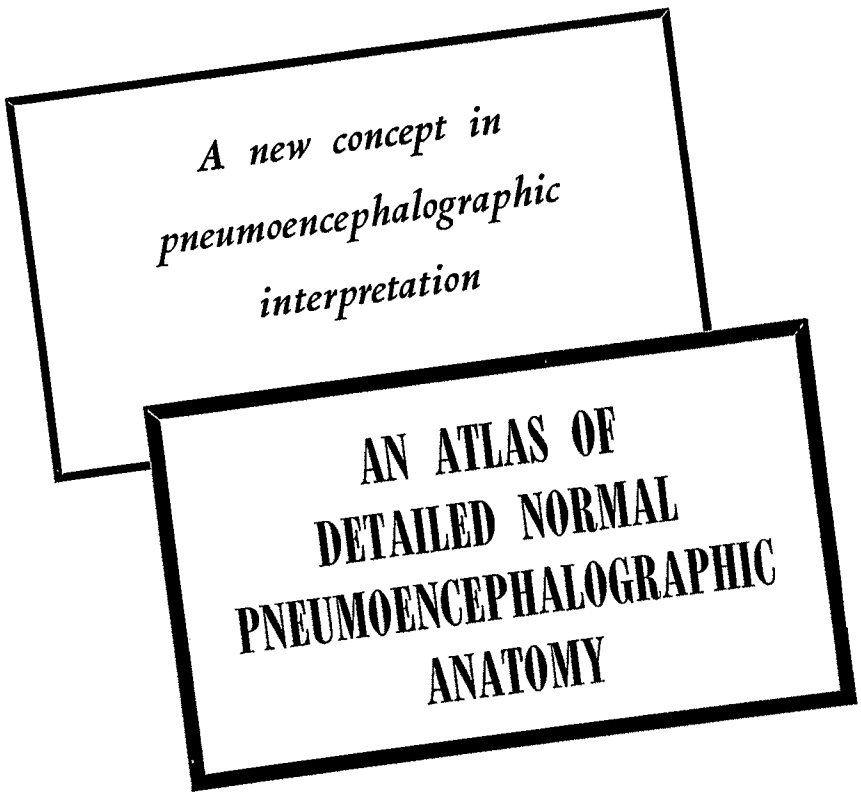
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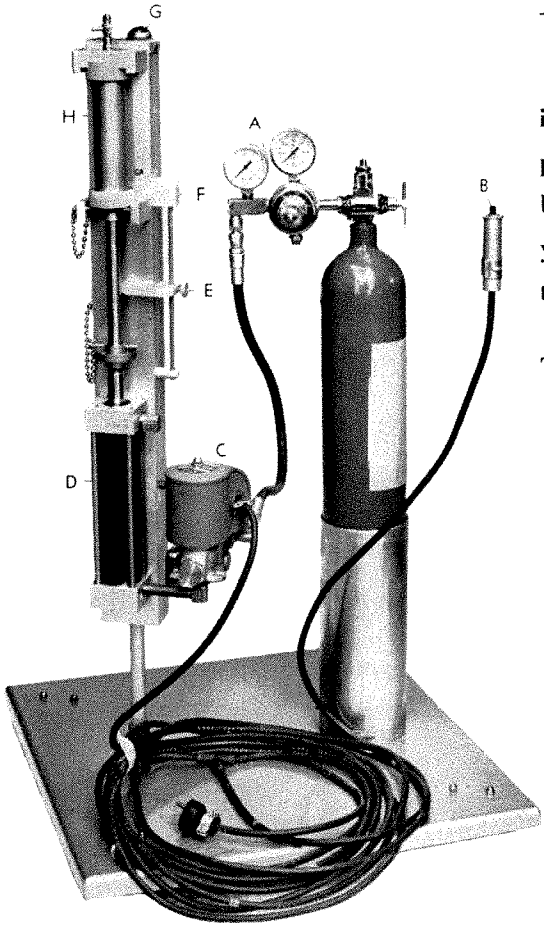
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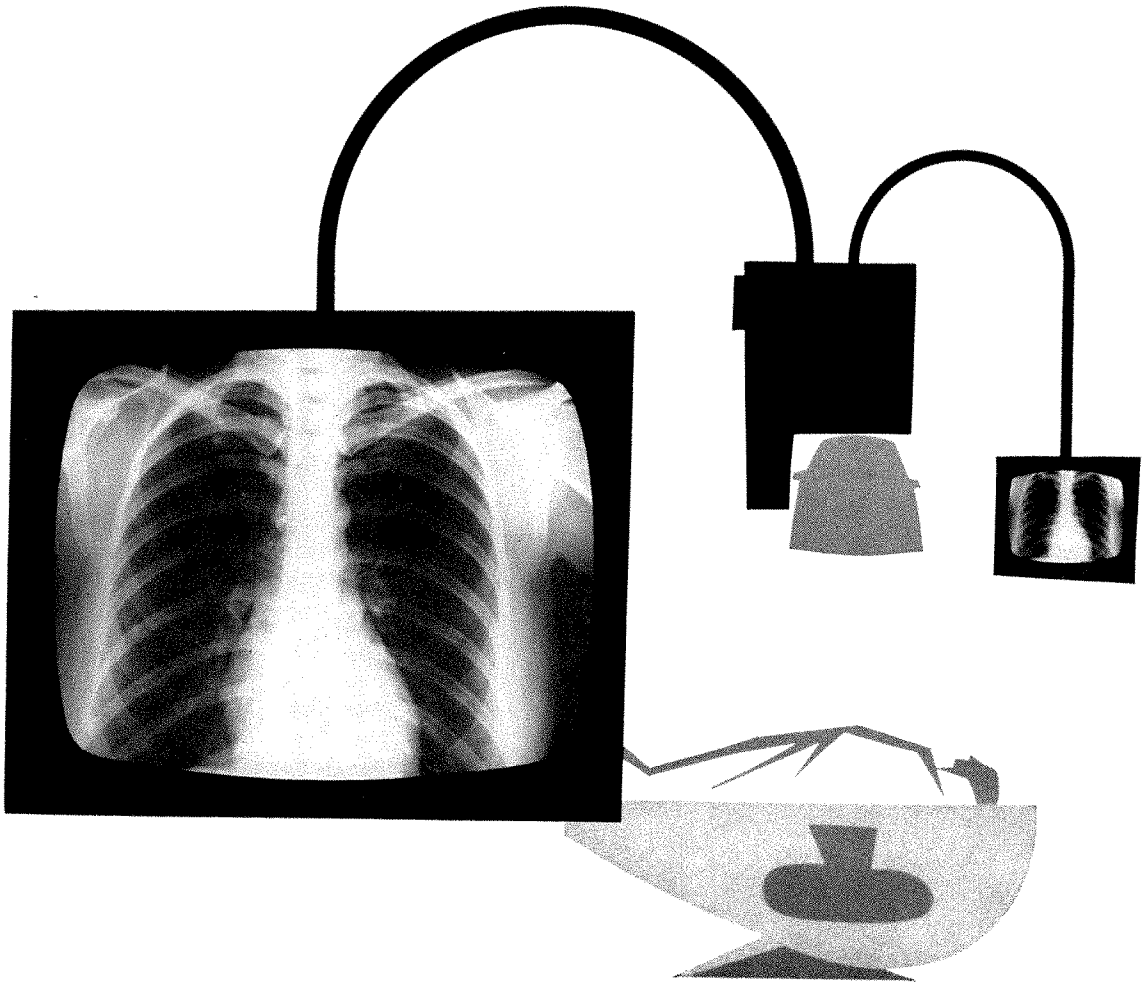
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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 86 SEPTEMBER, 1961 NO. 3

CINEROENTGENOGRAPHICALLY DEMONSTRATED CHANGES IN THE FETAL CIRCULATION OCCURRING AT BIRTH*

A SERENDIPIC COMMENTARY

By S. R. M. REYNOLDS, PH.D., D.Sc.
CHICAGO, ILLINOIS

It is an honor, however undeserved, for me to be asked to give the fifth W. Edward Chamberlain Lecture. It has not been my privilege to know Dr. Chamberlain until today, although the nature of his contributions to radiology reached even my distant ear some years ago upon his appointment to the Chair of Radiology at Temple University. My ear was distant not in space, for he became Professor of Radiology here at Temple University while I was still a graduate student at the University of Pennsylvania, just across the city. It was distant because my interest in basic medical science was far removed from the need for using radiologic techniques. How many endocrinologists, physiologists or embryologists have more than a nodding acquaintance with the uses of such tools for investigation? I was one of a majority, I am sure.

There came a time, however, when it became necessary for me in the course of my work to know how rapidly the blood flows through the pregnant uterus of the intact monkey. What better way to learn this than to propel some opaque medium at high speed through the linea alba, into the myometrium, and then observe how rapidly this was removed? To use isotopes would have left traces of radioactivity in tissues other than the myometrium, but roentgen rays offered the opportunity to localize the injected substance within the myometrium. We conducted a little study of this problem[4] and proved the feasibility of this technique (Fig. 1).

Even so, I did not even then learn first hand of Dr. Chamberlain. It really was not until I came to Temple University two years ago as one of a group of NIH consultants that I learned of the impact that he had made upon this institution. I found that here advanced radiologic techniques are used for research and teaching in the Departments of Physiology and Pharmacology as well as in Radiology. Here, these techniques are wisely and extensively employed. Of course, Dr. Stauffer and others have implemented such activities far beyond what Dr. Chamberlain did, but there is no doubt that Dr. Chamberlain has left here an intellectual legacy which is strong, growing and evident.

When Dr. Oppenheimer invited me to give the Chamberlain Lecture, I had serious doubts as to the wisdom of accepting the proffered honor. Upon consideration, however, I decided that perhaps my experience with serendipity in the field of radiology might make an interesting footnote in the annals of radiology, for it opened up avenues of investigation along which I have moved for ten

* From the Department of Anatomy, University of Illinois College of Medicine, Chicago, Illinois.
The W. Edward Chamberlain Lecture, delivered at Temple University School of Medicine, September 29, 1960.

years, and I presume I shall continue, with good fortune, to move along them for some few years to come.

SERENDIPITY, the gift of finding valuable and agreeable things not looked for, is a word coined by Walpole in allusion to a tale, *The Three Princes of Serendip*, who in their travels were always discovering by chance or sagacity, things they did not seek. My tale of serendipity goes back to my laboratory in Baltimore in 1948. A young doctor from Harvard came in and put on my desk some injection-corrosion specimens of the blood vessels of human placentas. He wanted help in developing a research project out of these specimens. In the course of our discussions, we noted something that no one else, especially any physiologist, apparently had observed. It was that, unlike virtually most other vascular beds, this one was unique. The cross-sectional areas of the veins were not appreciably different from the cross-sectional areas of the arteries that supplied the substance of the placenta. This being so, it readily suggested to us that the velocity with which the blood moves through the veins necessarily must be close to that at which the blood moves through the arteries.

From a physiologic standpoint this presented a dilemma. The blood in the arteries moves into the placenta under considerable pressure from the heart. But how about the pressure of blood in the veins? Where did the energy come from to propel the blood through the veins at as high a velocity, back toward the heart? It also raised the question of how much energy is lost against the vessel wall by blood flowing through a

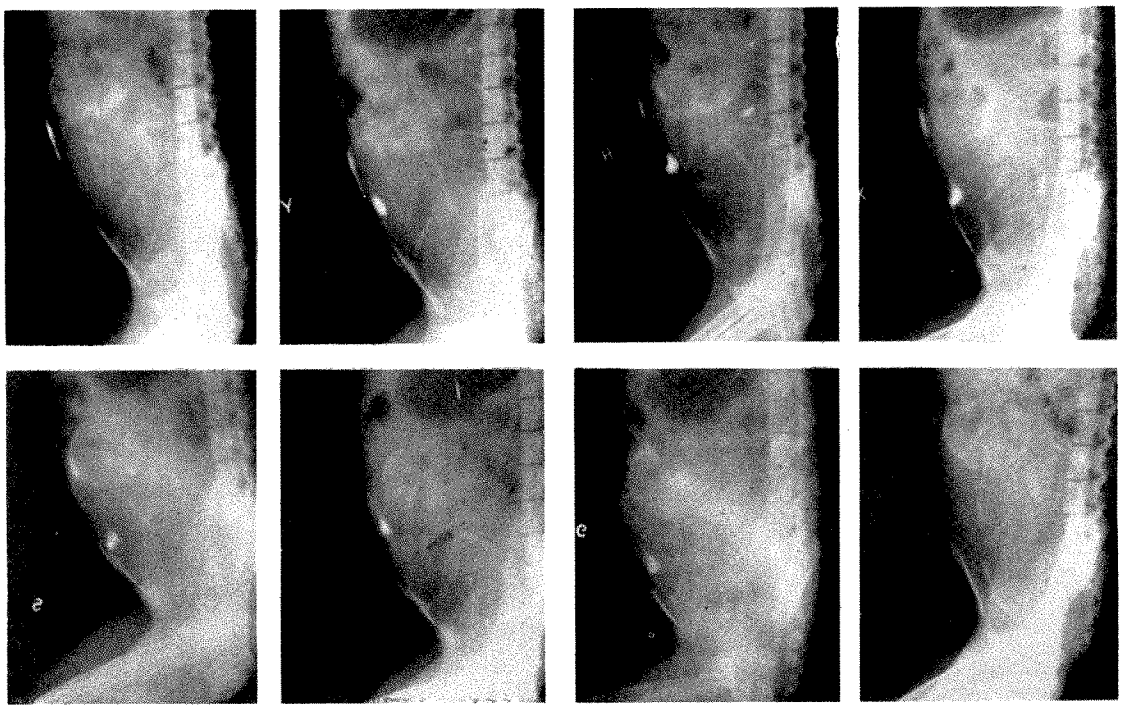


FIG. 1. Roentgenologic determination of an index to the blood circulation time in the intact uterus of a monkey pregnant 106 days. By jet hypospray, 1 ml. of diodrast is propelled through the linea alba into the myometrium. A residue from a previous injection of 1 ml. into the rectus abdominis muscle is seen in top left. Next right, 10 minutes later, the contrast medium had just reached the uterus. Succeeding roentgenograms were taken at regular intervals until both areas cleared. Twenty-four minutes were required for clearance in the the rectus abdominis, 34 minutes for the uterus. (Reproduced with permission from the *Proceedings of the Society for Experimental Biology and Medicine.*)

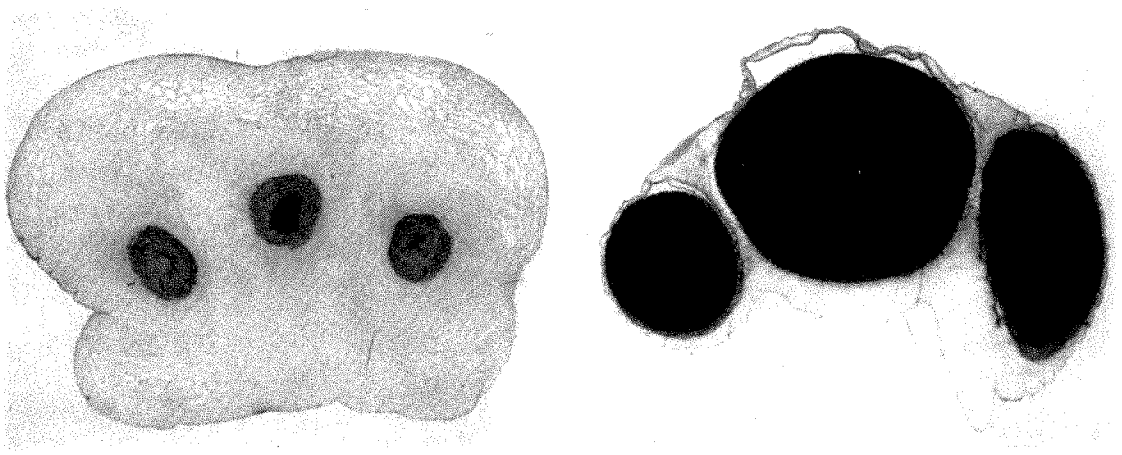


Fig. 2. Two sections of an umbilical cord. Left: conventional section with the two arteries and vein constricted and empty with much Wharton's jelly present. Right: section of the same cord taken with the infant held at the level of the placenta *in utero* and with two clamps placed simultaneously on the cord with a full pulsating beat. Note the large, distended vessels, the thin walls of the blood vessels and the thin layer of sheath about the cord. Wharton's jelly piles up as the cord shortens with vasoconstriction. (Reproduced with permission from *Contributions to Embryology*.)

vessel, and how much by internal dissipation of energy through the viscosity of the blood? Since the placenta seemed immune to *in vivo* experimental investigation, it occurred to me that perhaps this could be studied in the umbilical cord. The only difficulty was that histologists always study a cord after it is cut, with the blood drained out of it and the vessels constricted. Clearly, there had to be a way to determine the cross-sectional areas of distended and functioning umbilical arteries and veins.

I then went to a colleague in the Department of Obstetrics and asked him: Would you between the second and third stages of labor hold a baby at the level of the placenta, still retained within the uterus, and, with a full pulsating beat in the cord, put two clamps simultaneously at different points on the cord and then remove the distended segment?" This he did, whereupon I fixed the segment, with the clamps still on it. The result,[5] which was repeated many times, is shown in Figure 2. Here it was found that the combined areas of the two arteries were some 80 per cent of the area of the vein. So it was true that the blood in the vein moved only about 25 per cent slower than in the arteries. The functioning umbilical cord was clearly the tissue to be used to study the problem we had posed for ourselves, but where should we go from here?

I went to the Department of Sanitary Engineering at Johns Hopkins to talk to a colleague about our worries. He was a man noted for having successfully designed sewer systems for various towns and cities in the United States. Surely, I thought, he would know about fluid flows, and he could help me design an experiment to solve our problem.

Dr. Guyer was very helpful. What we would need, he said, was knowledge of the pressure drop along a length of blood vessel, the velocity of blood flow, the volume of blood flow per minute, and the amount and form of expansion of the blood vessel with each heart beat, since a blood vessel is nonrigid. We would need to know *all* these things simultaneously, and in one experiment!

There was but one place at that time where I could do this work—the Nuffield Institute for Medical Research, in Oxford, England. The reason for my selecting Nuffield Institute lay primarily in the fact that a book had been published a short while be-

fore by Barclay, Franklin and Prichard[2] entitled *The Foetal Circulation and Cardiovascular System, and the Changes They Undergo at Birth.* The approach in this now classic book had been entirely roentgenographic. They had taken serial roentgenograms at 2–5 images a second, and had learned much that was new as well as confirming much that had been known. I would remind you that, up to 1949, almost no one had produced truly good cineangiograms and there were no image amplifiers except in experimental stages. Moreover, at that time there were no laboratories where complicated animal experiments could be done using roentgen-ray techniques. This was what took me on a wandering trip to the British Isles, and points beyond.

But what did we discover that was valuable, agreeable and not looked for? I shall not tell you all that we learned. That would require many hours. Rather, I shall dwell on only a few facts that radiology revealed.

First, however, I must pay tribute to the radiologist who came to be my colleague. He is Dr. Gordon Ardran. Dr. Ardran considered the needs of my project and concluded that serial roentgenograms at 2–5 images per second would not suit our purposes. Instead, we would need true moving pictures, at better than 16 frames per second. With the limitation of little money, but with considerable resourcefulness, he bought a discarded moving picture projector from a local theater for the sum of ten pounds, or twenty-eight dollars. This, by ingenious adaptation, was made to take direct angiograms or cinefluorograms at a speed of 25 frames per second on 35 mm. film. So here, for almost the first time, true moving pictures of events could be taken by roentgen rays under highly experimental conditions. With this apparatus our work fell logically into two programs. One, of course, dealt with the umbilical cord. However, as we took cinefluorograms of the cord, we also obtained for the first time true cineangiograms of the fetal circulation. This, under the stimulus of Dr. Marjorie Prichard, who was a colleague, became our second area of study. We had no intention at the outset of engaging in fetal studies.

We learned much about blood flow in the umbilical cord. We found, for example, that if there is stream line flow (as there is in the larger vessels of almost the entire body) outside the ascending aorta and the arch of the aorta, the blood flows with surprisingly little loss of energy. In an umbilical vessel, there is only about a 2 per cent loss of energy in the system per centimeter of blood vessel. This means that, in the sheep fetus, about 90 per cent of the energy in the aorta is available to propel blood through the umbilical arteries.[9]

We[6] learned, too, that there is an advantage to an organism in having a blood vessel over a given diameter in order that it conduct blood efficiently (Fig. 3). For example, when a vessel has a diameter of 2–3 mm., the flow was 5–6 ml.[3] per second. When, however, the diameter was 3.4 or

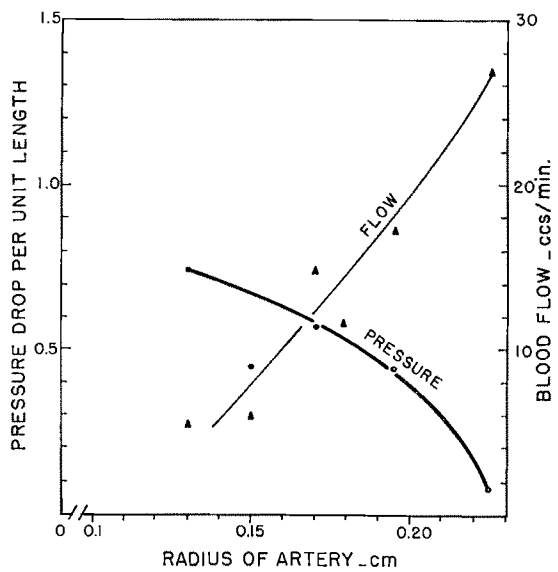


FIG. 3. Graph showing data obtained by cineangiography on the relation between the diameter of an umbilical artery and the flow of blood. The flow increases as the diameter increases; the pressure falls, as shown. (Flow is in ml./sec.) (Reproduced with permission from the *American Journal of Obstetrics and Gynecology.*)

4.0 mm., the flow was 15 and 17 ml.[3] per second, respectively.

The basis of these figures rests on the analysis of cineroentgenograms taken at 0.04 second. We could tell how many frames were required for the velocity-head of the opaque medium to traverse a given distance and we could, allowing for parallax, measure very well the diameter of the vessel studied. From these simple facts, unobtainable in any other way, we established these figures which any physiologist could have reasoned ought to apply, although he would not know what values to assign to the situation.

I have said that I went to Oxford to study hemodynamics in the umbilical cord. I have also noted that in so doing, we could not help but cineroentgenographically record the fetal circulation as well. Again we observed processes which we never anticipated might be available to us. When we saw the pictures projected, we were impressed by the fact, but hardly surprised, that we could see any major part of the fetal vascular tree. What could be simpler than to study how long a time was required for the opaque medium to pass from one part of the vascular tree to another? Thus, we could see it start toward the head in the areas supplying that region of the body, and we could see it return. Consequently, the cephalic circulation time could be measured. Similarly, the pulmonary circulation time was measurable, as were the renal, the portal, the hepatic, and the intestinal circulation times. We could even measure the length of time it took for the medium to pass down the aorta to one of the mesenteric arteries. We could—and did—measure the placental, and even the placental cotyledonary circulation times. In short, from cineangiofluorograms, we could learn for the first time what the regional circulation times in a fetus are.

Early in the study, Dr. Prichard, who had played an important role in the work on the fetal circulation, said, "I would like to see what happens when we give the fetus artificial respiration." Now, to put this in perspective, it is necessary to state what Barclay, Franklin and Prichard[2] had established earlier.

They had found that the unique features of the fetal circulation undergo profound changes and in an apparent sequence upon establishment of respiratory movements. One of these features is closure of the ductus venosus, that structure lying deep within the recesses of the liver which is the main connection between the end of the umbilical vein and the inferior vena cava. It is so well protected by soft and blood-loaded tissue that it can only be studied functionally by roentgenography. Closure of this sphincter, they postulated, prevented hemorrhage through the torn umbilical cord after birth, for most mammals at birth are unattended by an obstetrician who clamps or ties the severed umbilical cord.

The second significant change at the time of birth is closing of the ductus arteriosus which, as these authors demonstrated so elegantly, interposes a block between the pulmonary artery and the aorta after establishment of breathing, and so directs all the blood from the right heart into the lungs. The third essential change is closure of the foramen ovale, the opening in the interatrial septum that serves during fetal life to admit blood from the right to the left side of the heart, thus bypassing the fetal lungs. In their study, it is never precisely stated what the order or sequence of closure of these structures is. It is here that, as we wandered through one experimental procedure after another, we uncovered a series of related facts of immeasurable importance to our understanding the nature of the fetal circulation and the changes it undergoes at the time of birth. We will look at these, one by one.

First, the ductus venosus. To understand this, I must narrate my talk with the engineer, Dr. Guyer. When I talked to him, he asked about the pressure in the umbilical vein (which I knew to be high) and how the vein ended in the liver. I sketched for him the umbilical recess in the liver, showing

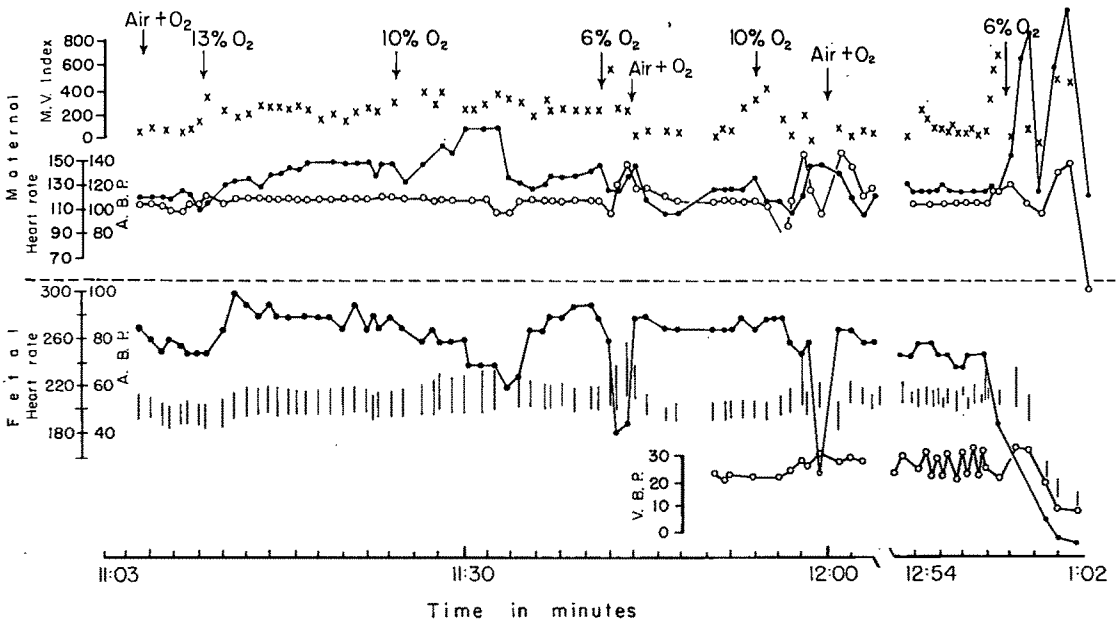


FIG. 4. Graph showing the effect of umbilical vein pressure on the welfare of the ewe fetus *in utero*, with the membranes intact, during severe hypoxia. Events in maternal ewe (top): respiratory minute volume index, x; heart rate, o; mean carotid blood pressure, •; in the fetus (bottom): umbilical artery pulse pressure, |||; umbilical vein pressure, —. Note that on 13 per cent and 10 per cent O_2 is ewe's inspired air; fetal heart rate was variable as arterial pulse pressure improved. Venous pressure held constant at 20–28 mm. Hg. On the second exposure to 6 per cent O_2, maternal hemodynamic disturbances occurred and the heart rate fell, as before; venous blood pressure fell (with probable closing of the umbilical vein) and then arterial pressure fell. The fetus died.

the ductus venosus as a wide, open structure passing into the inferior vena cava, and many small tributaries going off into the substance of the liver. He put his pencil on the ductus venosus and said, "Where is the valve here that controls the pressure back here in the umbilical vein?" I said, "There is none." He said there had to be. Then, recalling that there is a sphincter in the ductus venosus as was shown both roentgenographically and anatomically by Barclay, Franklin and Prichard,[2] he told me that hemodynamic pulsations in the umbilical arteries exerted intermittent pressures on the distended vein, and so "milked" the blood in a forward direction toward the heart. It was, he said, a pulsameter pump such as is used in the chemical, petroleum and other industries. Now this was a new idea, that the sphincter served a vital role in the maintenance of blood flow from the placenta to the fetus, instead of merely being a stopcock to shut off a retrograde flow of blood afer the organism was born. What, in our wandering, did we find?

To be brief, we found, as had Barclay, Franklin and Prichard,[2] that the sphincter may be either closed or open during fetal life. In one fetus only in which experiments were done, it was found that the sphincter could be made to open by progressively reducing the venous return from the placenta. Before leaving this subject, it should be said that later studies in recent years have shown that if umbilical vein pressure becomes low, a fetus becomes moribund and dies. If, however, it remains high, the fetus withstands stress and is very vigorous[10] (Fig. 4).

The most dramatic circulatory change in the fetus upon aeration of the fetal lungs is in the quality of the hemodynamic change in the lung itself. It is the first and largest change to take place. Dawes[3] has shown

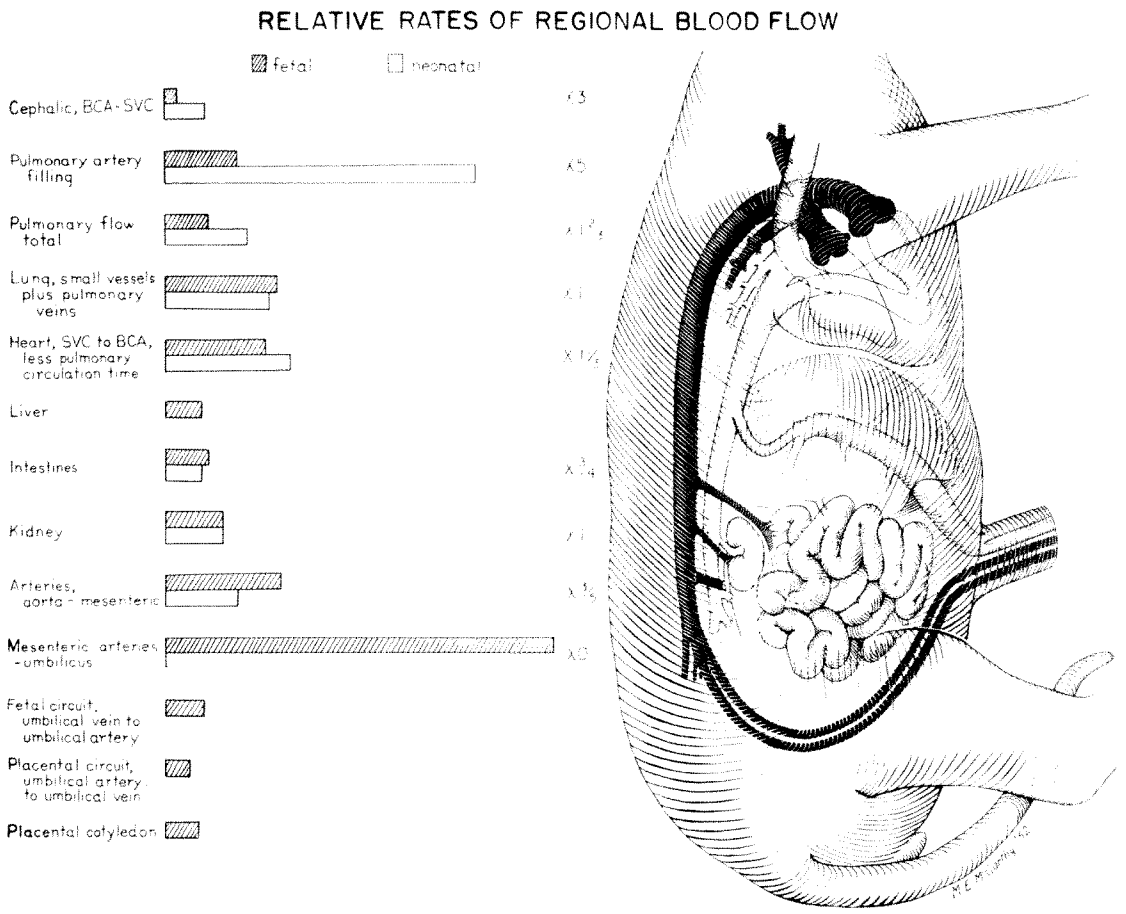


Fig. 5. Roentgenographically determined circulation times for opaque medium passing from one part of the circulatory tree to another in the fetus and breathing lamb. Cineangiograms taken at 25 frames per second permitted noting, from frames at 0.04 second apart, the transit time of the opaque medium. The spread of circulation is the reciprocal of the circulation time. The bar graphs show (fetus, striped bars; breathing lamb, open bars) that the cephalic circulation is 3 times faster after birth; the pulmonary circulation (arterial transit time) is 5 times faster, and so on. (Reproduced with permission from *Contributions to Embryology*.)

that during fetal life only about 13–15 per cent of the fetal cardiac output goes to the lung. Ardran *et al.*[1] demonstrated that the pulmonary circulation in the fetus is the slowest regional circulation (Fig. 5); however, upon aeration of the fetal lung, it becomes the fastest. This is associated with the group of physiologic changes detailed in Table I.[7]

The cineroentgenographic demonstration of this pulmonary change, which was missed in the serial film technique, required that the anatomic nature of the fetal and

Table I

PHYSIOLOGIC CIRCULATORY CHANGES

Physiologic Condition	Fetal	Neonatal
Pulmonary Artery Pressure	++++	+
Pulmonary Peripheral Resistance	++++	+
Rate of Pulmonary Blood Flow	++	+++
Pulmonary Blood Volume	+	++-+++

neonatal circulations be established. This was done in 1956,[7] although to describe these results here would carry us far afield from our present theme. However, I would point out that the roentgenographic technique led inadvertently to a new line of anatomic and physiologic experiments on the nature of fetal physiology and its bearing on postnatal life.

Before concluding, some specific results of the cineroentgenographic observations on the fetal circulation should be pointed out. Careful analysis of the films showed that in the fetus circulation is slowest through the head and is slow in the lungs, liver, intestines and kidneys. The fastest and most copious flow is to and through the placenta. Upon aeration of the lungs, there is a two and one-halffold increase in the cephalic circulation, a fourfold increase through the lungs, and a complete cessation, of course, to the placenta. The flow through the heart, kidneys and intestines is unchanged. In short, Nature has contrived to have the most rapid and abundant flow of blood through the organs of respiration in both the fetus and the neonatal animal.[8] This is not unexpected, but it required cineangiography to prove the point.

I would be remiss if I did not reiterate that these initial observations have led to a succession of completed studies on fetal homeostasis, fetal distress and the life of the fetus within the uterus, with the membranes and fluid surrounding the fetus still intact. These studies have led to others which are still in active progress. It is no exaggeration to say that the casual observation of the fetus by cinefluoroangiography in the course of another study has been the most important means whereby a whole array of heretofore unsuspected problems in physiology and anatomy have come into view. In this case, serendipity has served us well.

Department of Anatomy
University of Illinois College of Medicine
Chicago, Illinois

REFERENCES

1. ARDRAN, G. M., DAWES, G. S., PRICHARD, M. M. L., REYNOLDS, S. R. M., and WYATT, D. G. Effect of ventilation of foetal lungs upon pulmonary circulation. *J. Physiol.*, 1952, *118*, 12–22.
2. BARCLAY, A. E., FRANKLIN, K. J., and PRICHARD, M. M. L. The Foetal Circulation. Charles C Thomas, Publisher, Springfield, Ill., 1945.
3. DAWES, G. S. In: Proc. Cold Spring Harbor Symposium. Mammalian fetus: physiological aspects of development. 1955, V. 19.
4. GILLESPIE, E. C., and REYNOLDS, S. R. M. Uterine circulation time in pregnant primate, with uterus and abdomen intact. *Proc. Soc. Exper. Biol. & Med.*, 1949, *70*, 721–724.
5. REYNOLDS, S. R. M. Proportion of Wharton's jelly in umbilical cord in relation to distention of umbilical arteries and vein, with observations on folds of Hoboken. *Anat. Rec.*, 1952, *113*, 365–377.
6. REYNOLDS, S. R. M. Circulatory adaptations to birth and their clinical implications. *Am. J. Obst. & Gynec.*, 1955, *70*, 148–161.
7. REYNOLDS, S. R. M. Fetal and neonatal pulmonary vasculature in guinea pig in relation to hemodynamic changes at birth. *Am. J. Anat.*, 1956, *98*, 97–115.
8. REYNOLDS, S. R. M., ARDRAN, G. M., and PRICHARD, M. M. L. Observations on regional circulation times in lamb under fetal and neonatal conditions. *Contrib. Embryology*, 1954, *35*, 73–92.
9. REYNOLDS, S. R. M., LIGHT, F. W., JR., ARDRAN, G. M., and PRICHARD, M. M. L. Qualitative nature of pulsatile flow in umbilical blood vessels, with observations on flow in aorta. *Bull. Johns Hopkins Hosp.*, 1952, *91*, 83–104.
10. REYNOLDS, S. R. M., and PAUL, W. M. Relation of bradycardia and blood pressure of fetal lamb *in utero* to mild and severe hypoxia. *Am. J. Physiol.*, 1958, *193*, 249–256.

VOL. 86, NO. 3

THE ROENTGENOLOGIC DIAGNOSIS OF FETAL HYDROPS*

By PAUL A. BISHOP, M.D.
PHILADELPHIA, PENNSYLVANIA

IN A paper entitled "The Fallibility of Radiological Diagnosis of Erythroblastosis Foetalis," McClure Browne[9] states, "A radiograph of the foetus *in utero* showing the Buddha position and a halo around the skull is usually accepted as diagnostic of erythroblastosis foetalis. These signs were first described by Hellman and Irving (1938). Javert (1942) laid special emphasis on the value of the Buddha position. Ritvo, Shauffer and Krosnick (1949) claim that the signs enable a definite diagnosis of erythroblastosis foetalis to be made. Hartley (1948), however, regards them with distrust. I, too, have sometimes found these signs to be misleading, and suggest that they be valued only together with all evidence of severe foetal damage." He continues with reports of 2 cases of pregnancies producing normal babies in which an incorrect diagnosis of fetal hydrops had been based on the halo and Buddha signs.

Because of the apparent unreliability of the halo sign noted by others as well as ourselves and the diagnostic inadequacy of the Buddha position alone, we feel justified in presenting our experience with both direct and indirect roentgen signs of fetal edema. In so doing we add 4 cases to the 2 we reported in 1953.[1]

Modern methods in the care of premature infants and the spectacular results of replacement transfusion techniques make early diagnosis of this condition of great practical importance. One of our cases (Case 5) was not suspected clinically. The roentgenologic diagnosis of fetal hydrops (Fig. 8, *A* and *B*) stimulated prompt action. Cesarean section followed by immediate and repeated replacement transfusions resulted in a vigorous baby which continues to thrive. Armed with this procedure we can no longer be content to wait for "evidence of severe fetal damage" as suggested by McClure Browne.[9] Ritvo *et al.*[11] state, "as with other rare and unusual conditions, the diagnosis will be made only if the manifestations are clearly understood, constantly borne in mind and painstakingly sought for in every instance. The importance of early diagnosis in planning therapy and making a proper prognosis is self evident."

Erythroblastosis fetalis is a hemolytic clinico-pathologic syndrome of offspring resulting from conception between an Rh negative woman and an Rh positive man. The first born child is seldom affected unless the mother has previously received a transfusion of Rh positive blood. Fifteen per cent of white mothers are said to lack Rh agglutinogens and are therefore termed Rh negative. Sensitization of these mothers by the Rh positive red blood cells of the fetus causes the formation of an anti-Rh agglutinin. Erythroblastosis fetalis develops when this diffusible substance passes into the circulation of the fetus. With each succeeding pregnancy the probability of producing a baby with hemolytic disease is increased. Pickles[10] has stated that the chances of an Rh negative mother becoming immunized during the first pregnancy and therefore producing an infant with hemolytic disease are 1 in 1,000. For the second and third pregnancies the chances rise to 1 in 40 and increase to about 1 in 15 for the fifth pregnancy. Javert[7] found that the incidence of erythroblastosis fetalis after its initial appearance is nearly 50 per cent and if the first born had the disease the incidence is nearly 100 per cent. Hellman

* From the Department of Radiology, Pennsylvania Hospital, Philadelphia, Pennsylvania.
Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

and Irving[5] estimate that fetal hydrops occurs once in 2,000 deliveries.

CLINICAL AND PATHOLOGIC FEATURES

The clinical manifestations of erythroblastosis fetalis fall into three groups. In order of increasing severity these are: (a) congenital anemia; (b) icterus gravis; and (c) fetal hydrops. In the mildest form, congenital anemia, the diagnosis is suspected by the pallor of the infant and confirmed by blood studies.

Icterus gravis is characterized by jaundice of varying degree that may be present at birth or may not develop for several days. The jaundice is usually progressive and is accompanied by progressive anemia.

The most severe form of erythroblastosis fetalis, fetal hydrops, is characterized by marked generalized edema of the soft parts, associated with accumulation of fluid in the pericardial, pleural and peritoneal cavities, and enlargement of the heart, liver and spleen (Fig. 1, *A* and *B*, Case 4). The blood picture is that of marked anemia associated with immature red blood cells. The placenta is large, thickened and edematous (Fig. 1 *C*, Case 4). As a result of the increased size of the fetus and the placenta, the uterine enlargement is found to be out of proportion to the duration of the pregnancy and hydramnios is generally suspected clinically. Javert[7] reports that in the 16 cases which he studied, "clinical hydramnios" was due to the combined excessive size and weight of the placenta and the infant which averaged 4,674 gm. (10.2 pounds). The placenta averaged 1,254 gm., the fetus 3,420 gm. This is a 1 to 3 ratio in contrast to the normal 1 to 6 ratio. In 5 of our cases there was a clinical diagnosis of hydramnios.

Bone changes are quite constant, occurring in the anemic and icteric forms as well as in fetal hydrops. Follis *et al.*[3] and Janus and Dietz[6] found marked increase in bone density on postnatal roentgen studies as well as on microscopic postmortem studies. There was an increase in the number and thickness of trabeculae, due apparently to lack of destruction of the calcified cartilaginous matrix substance which was then covered with a thick layer of new bone. The medullary spaces were narrowed or obliterated. In long bones a transverse zone of decreased density close to the junction of the shaft and cartilage was frequently present. This is the result of transient disturbance of endochrondal bone formation and not distinguishable from other prenatal interruptions of bone formation.

INDICATIONS AND TECHNIQUE OF ROENTGEN EXAMINATION

Indications for roentgenologic examination arise during the last two months when a pregnant woman is known to be Rh negative or has had a previous erythroblastotic baby. Hellman and Irving[5] recommend that such patients be examined every two weeks during the last two months of pregnancy. Ritvo *et al.*[11] advocate frequent reexaminations to detect early changes that might otherwise go unrecognized, the first, ten to fifteen weeks prior to term and thereafter at three to six week intervals.

Clinical suspicion of hydramnios is generally considered as a mandatory indication for roentgen examination due to the frequency with which fetal anomalies occur with this condition. Fetal hydrops must be added to the list of abnormalities to be thought of and searched for in such examinations, since uterine enlargement out of proportion to the fetal age in erythroblastosis cannot be differentiated clinically from true hydramnios.

Javert[7] found a high incidence of preeclamptic toxemia in mothers of hydrops cases (31 per cent of 16 cases). Two of our 6 cases were toxic (Table 1). Cases showing symptoms of toxemia when associated with hydramnios should be investigated roentgenographically.

Painless bleeding is a frequent symptom, occurring in 3 of our 6 cases (Table 1). The possibility of fetal hydrops must be considered in every case examined roentgenographically for placenta previa.

The technique of the examination is

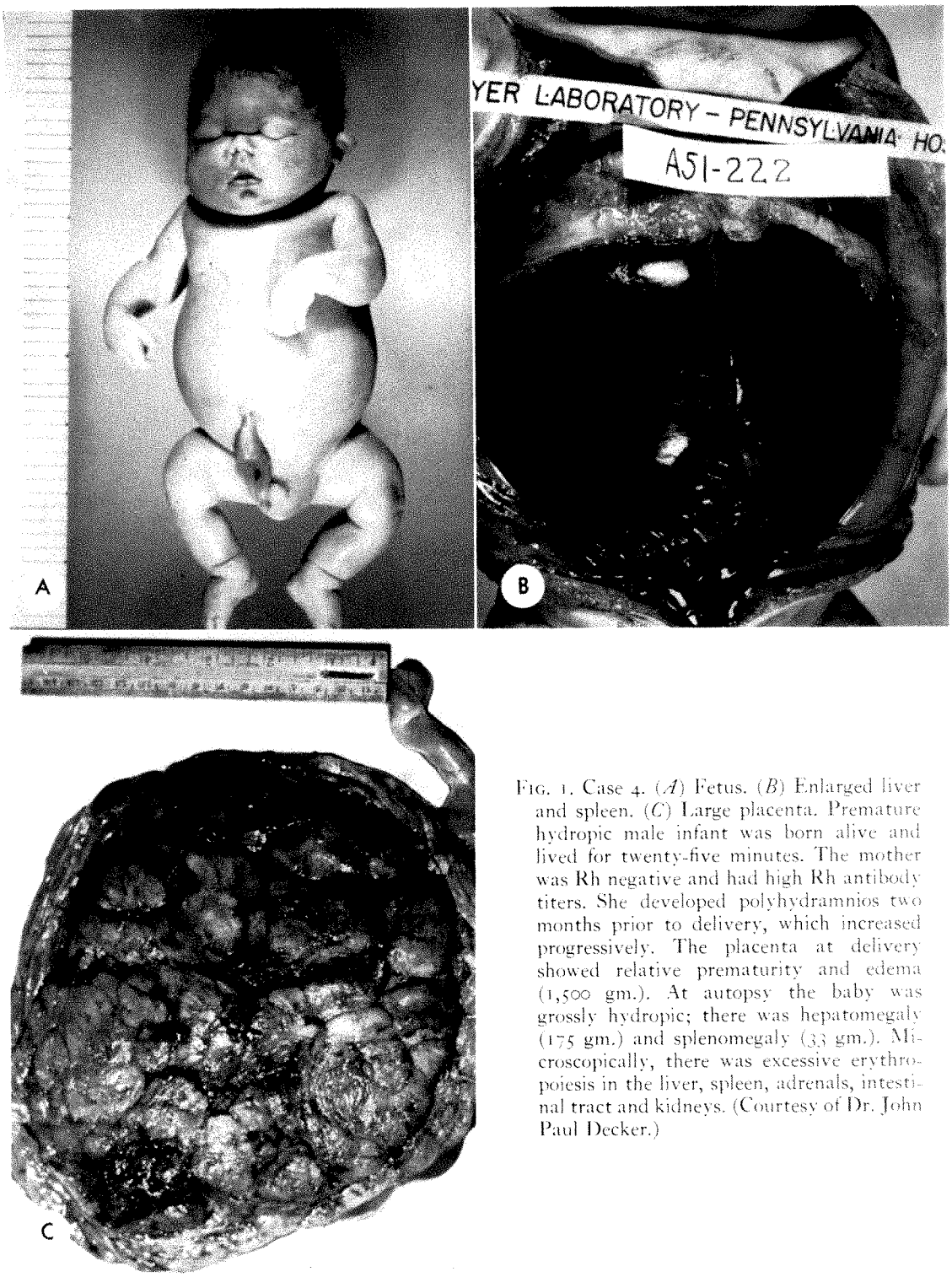


FIG. 1. Case 4. (*A*) Fetus. (*B*) Enlarged liver and spleen. (*C*) Large placenta. Premature hydropic male infant was born alive and lived for twenty-five minutes. The mother was Rh negative and had high Rh antibody titers. She developed polyhydramnios two months prior to delivery, which increased progressively. The placenta at delivery showed relative prematurity and edema (1,500 gm.). At autopsy the baby was grossly hydropic; there was hepatomegaly (175 gm.) and splenomegaly (33 gm.). Microscopically, there was excessive erythropoiesis in the liver, spleen, adrenals, intestinal tract and kidneys. (Courtesy of Dr. John Paul Decker.)

TABLE I

CLINICAL SUMMARY

Case	Hydramnios	Bleeding	Toxemia	Fetal Death	Rh	Previous Babies
1 J.W.	+	−	+	+	Neg.	Icterus gravis, Anemia
2 M.S.	+	+	−	+	Neg.	3 Normal
3 A.Y.	+	−	+	+	Neg.	2 Fetal hydrops
4 G.McA.	+	+	−	Alive 25 min.	Neg.	1 Erythroblastosis fetalis
5 C.C.	+	+	−	Living, well	Anti C factor	1 Normal 1 Icterus gravis
6 A.R.	−	−	−	Alive 2 hrs.	Neg.	1 Fetal hydrops

simple. Anteroposterior and lateral roentgenograms are made with the patient lying on a Bucky diaphragm table. High speed films and intensifying screens are used in an effort to reduce the radiation dose to both mother and child to a safe minimum. The dangers of fetal damage from repeated exposures in doubtful cases must be carefully weighed against the importance of each examination. The roentgenographic technique is of the quality used in urologic studies. Roentgenograms must be made in a minimum of time, thereby avoiding primary motion of the fetus and secondary motion of the mother. Fine detail is essential; retakes are undesirable.

ROENTGEN FINDINGS

In 1934 Snow and Powell[13] described a "black line" outlining the neck, trunk and extremities on roentgenograms of the gravid abdomen made by a soft tissue technique. This black line (Fig. 2) is due to the greater penetrability by roentgen rays of the fat containing subcutaneous tissues.[13] It was noted that it was widest over the buttocks, a notorious depository of fat, and absent over the cranium because there is no subcutaneous fat layer in the scalp. This black line, which outlines the trunk and extremities of the fetus, has become an important landmark in roentgenologic studies for localization of the placenta as well as in the diagnosis of short umbilical cord.[1] The obliteration of this fat line, secondary to edema of the soft tissues, has become our most reliable sign in the diagnosis of fetal hydrops. This corresponds to other time-proven roentgenologic changes of diagnostic importance, such as the disappearance of the shadows of the perirenal and psoas muscle fat pads in cases of retroperitoneal inflammations or tumor infiltration and the obliteration of the properitoneal fat line in patients with ascites. It should be emphasized that the fetal fat line is not obliterated when the fetus is surrounded by the excessive amount of amniotic fluid in hydramnios (Fig. 3).

In 4 of our cases prenatal roentgenograms showed the fetal fat line to be completely obliterated. In Case 5 and 6, both live fetuses, the fat line was seen very faintly along the back where the edema was less marked; it was completely obliterated over the abdomen and the extremities (Table II). In a review of the literature, roentgenograms of one case in Javert's[7] series and in 3 reported by Ritvo *et al.*[11] show the shadow of the fat line to be ab-

sent, although this finding is not mentioned in their texts. McClure Browne[9] presents beautiful illustrations of a normal case in which an incorrect diagnosis of fetal hydrops was made on the basis of the Buddha position and the halo sign. The fat line of the baby is shown with clarity. The application of our diagnostic standards would have ruled out a diagnosis of fetal hydrops in this case.

The position of the fetus in relation to the uterus is of diagnostic importance. The fetus usually lies with its back in close contact to the uterus while anteriorly the distance to the uterine wall is much greater than normal. This is due to the thickened, edematous placenta and the distended abdomen of the fetus.

The posture of the fetus is noteworthy. The ribs are elevated and in a more or less horizontal position due to the enlarged heart, the pleural fluid and upward pressure of the distended abdomen. The arms are spread outward and lie well anteriorly, displaced from the normal position of close contact with the trunk because of the soft tissue edema. The legs, likewise, assume an abnormal "frog like" position, the so-called Buddha sign. They are flexed at the knees and abducted at the hips. The extremities of a normal fetus may and frequently do assume a similar posture. This appearance, however, is important in combination with other signs of fetal edema

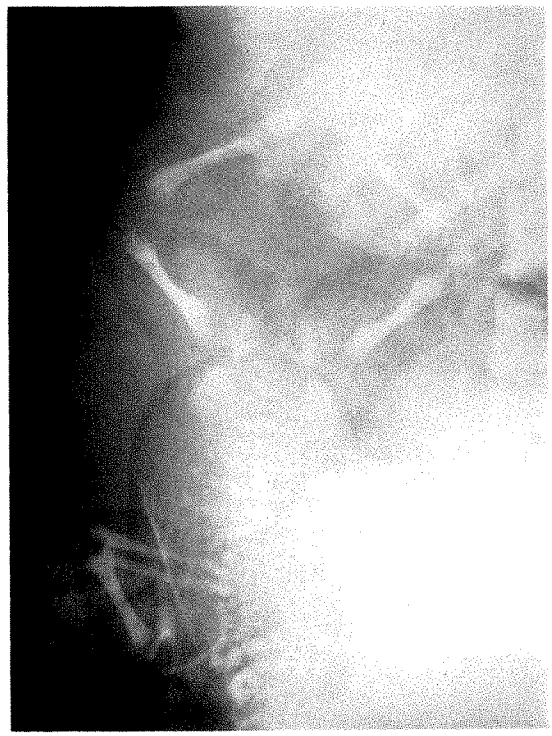


FIG. 2. Fat line or "black line" of subcutaneous fat surrounding the trunk and extremities of a normal fetus.

TABLE II

ROENTGEN SUMMARY

Case	Fetal Position	Cranial Halo	Fetal Fat Line	Bones					
				Density	Position				Cranial Bones
					Ribs	Arms	Legs	Spine	
1 J.W.	Breech	No	Completely obliterated	Moderate increase	Elevated	Out and elevated	Flexed 90° abducted	Straight	Overlapping
2 M.S.	Breech	No	Completely obliterated	Slight increase	Elevated	Out and elevated	Flexed 135° abducted	Straight	Overlapping
3 A.Y.	Transverse	No	Completely obliterated	Marked increase	Elevated	Out and elevated	Flexed 135° abducted	Increased curve	Overlapping
4 G.McA.	Vertex	No	Completely obliterated	Normal	Elevated	?	?	Straight	Normal
5 C.C.	Vertex	No	Partly obliterated	Normal	Elevated	Out	Flexed 90°	Normal curve	Normal
6 A.R.	Vertex	No	Partly obliterated	Increased	Elevated	Out	Flexed abducted	Normal curve	Normal

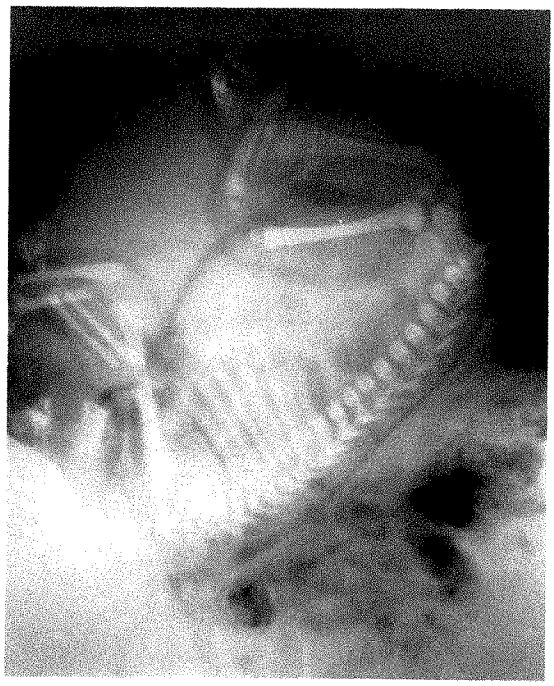


FIG. 3. "Black line" in a case of true hydramnios (ancephalic monster).

and enlargement of the abdomen and thorax.

The posture of the spine is of interest. Samuel and Cohen[12] state that the spine loses its normal curvature as a result of the enlargement of the liver and the spleen and ascites, but add that other intra-abdominal masses produce the same effect. Our findings are not uniform. In 3 or our cases, in which the fetus was dead and macerated, and lying in the breech or vertex position (Fig. 4, *A* and *B;* 5, *A* and *B;* and 7; Case 1, 2 and 4), there was complete absence of the normal spinal curve; 2 cases in vertex position (Fig. 8, *A* and *B;* and 9; Case 5 and 6), both alive, had normal spinal curves; another dead fetus (Fig. 6, *A* and *B*, Case 3) in a transverse lie had an exaggerated flexion curvature of the spine.

The halo sign has not been of diagnostic value in our series. Hellman and Irving[5] in their original description said, "The edema is seen in roentgenograms as a thickening

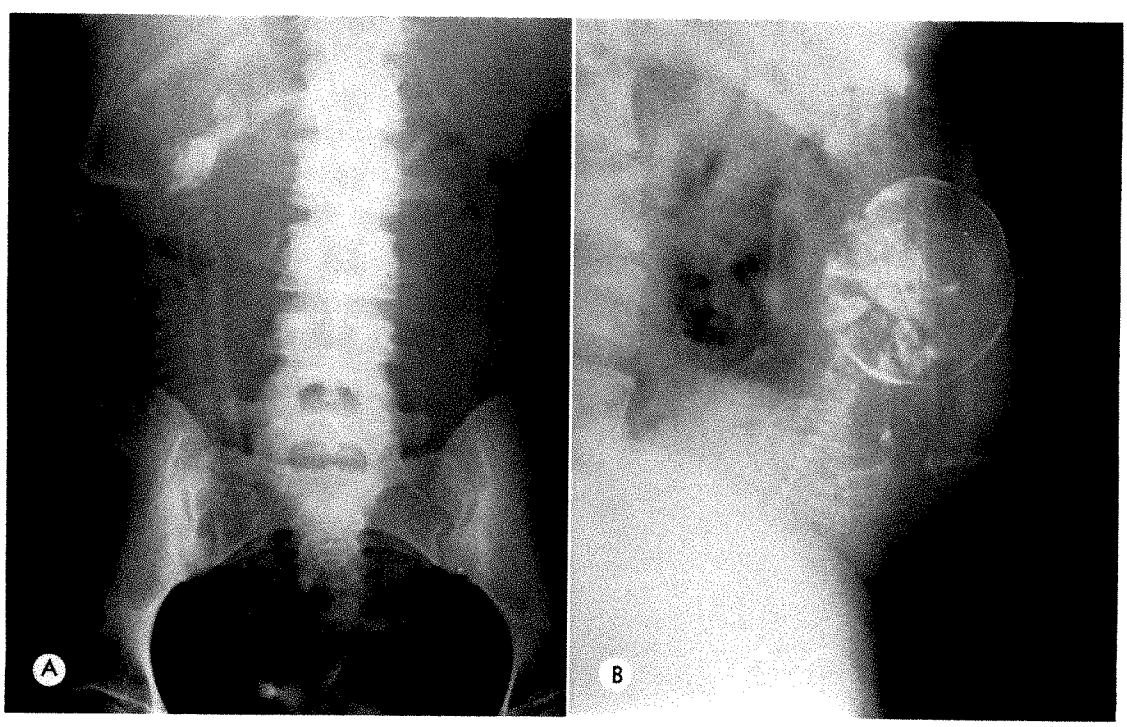


FIG. 4. Case 1. (*A* and *B*) The mother was a twenty-three year old white female, para II. The first baby, jaundiced at birth, is well at three years of age. During the second pregnancy, the patient developed signs of toxemia. Roentgenograms revealed a dead fetus, absence of a fetal fat line and elevated ribs of the fetus. The blood of the father was Rh positive; the blood of the mother was Rh negative and the anti Rh factor was present. Cesarean section revealed a dead fetus with fetal hydrops.

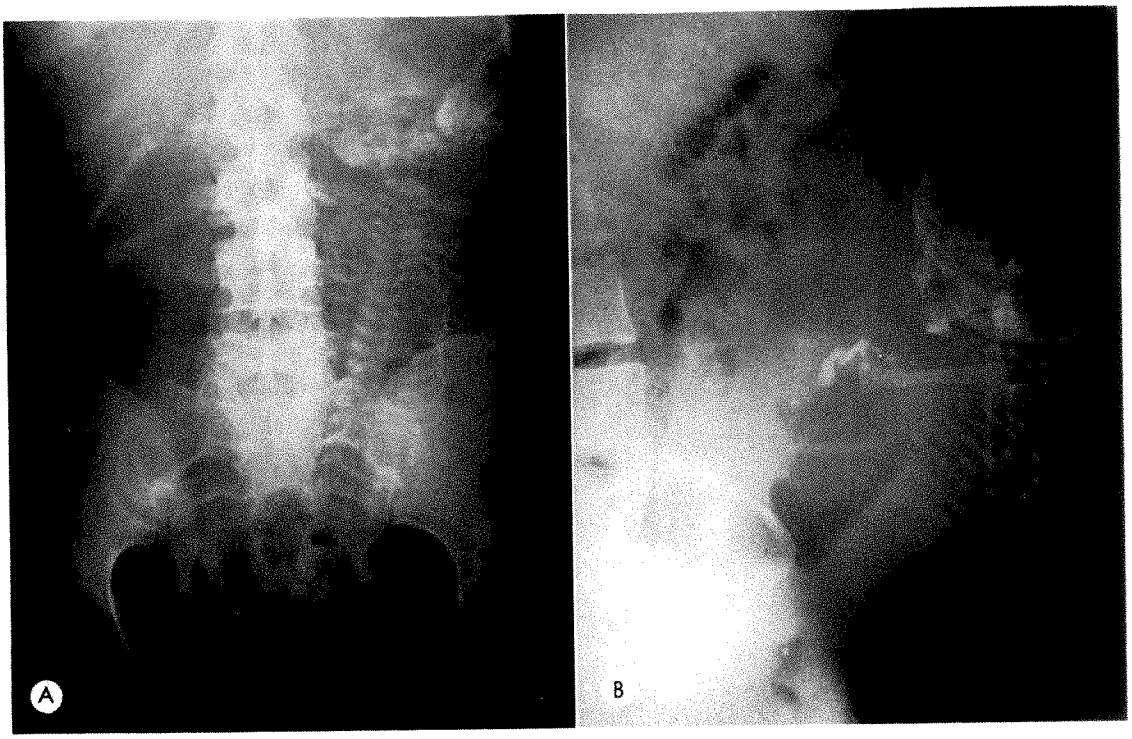


FIG. 5. Case 2. (*A* and *B*) The mother was a thirty-five year old white female, para IV. She had 3 norma pregnancies in close succession. The present pregnancy was normal up to thirty-eight weeks, when fetal movements and heart sounds were absent. Two days later profuse vaginal bleeding occurred. Roentgen examination for placenta previa revealed signs of fetal hydrops. Four days later a macerated dead fetus with fetal hydrops was delivered. The mother's blood was Rh negative; it had been Rh positive during the 3 previous pregnancies.

and increased density of soft parts. It is so striking in the scalp that a definite corona is seen surrounding the skull." They also speak of it as a "dark corona." A dark shadow could result only from tissues of decreased roentgenographic density similar to fat. Javert,[7] on the other hand, illustrates the halo sign in what appears to be a retouched reproduction in which the edematous scalp casts a shadow that is lighter than the surrounding soft tissues. The fact that such prominent proponents of the halo sign present it in opposite densities is confusing, to say the least. Postnatal roentgenograms reveal a thickened edematous scalp which casts a shadow of the same density as the other edematous soft tissues. That this "halo" is said to be present only in vertex presentations is also difficult to rationalize. We have observed on roentgenograms of many normal obstetrical cases with vertex presentation the shadows of the soft tissue planes of a partially filled and compressed maternal bladder cause an appearance strikingly similar to a "halo." McClure Browne[9] presents illustrations of 2 normal cases in which shadows about the head were interpreted as the halo sign. One of these is typical of compression of the maternal bladder by the presenting fetal head; the other appears to be the double bone shadow of the vertex due, I believe, to overlapping shadows of the parietal bones. We are not convinced that the halo sign is of diagnostic value. The mechanism of its production is not clear. Illustrations of it in the literature are confusing. It was not observed in any of our cases.

The increased bone density of the fetus likewise was not a striking feature in the prenatal roentgenograms in our series. Only in Case 3 was it of diagnostic significance. It was probably present in other cases but masked by the increased density

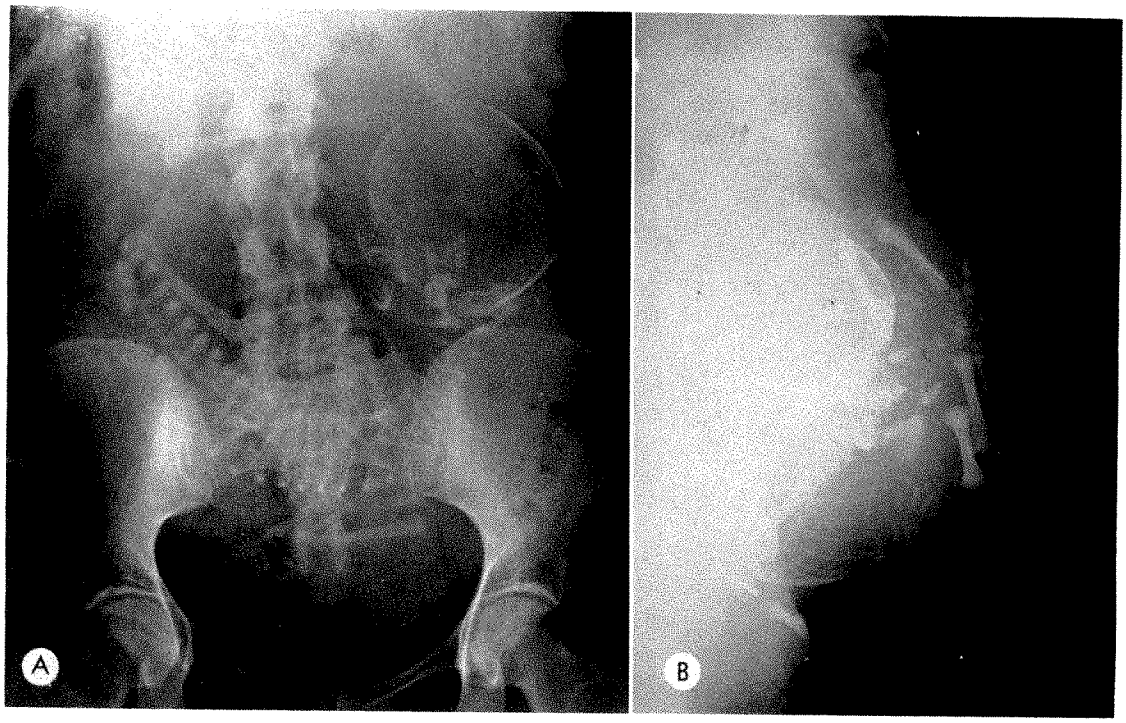


FIG. 6. Case 3. (*A* and *B*) The mother was a thirty-one year old white female, para III. She was known to have an Rh negative factor and had lost 2 previous pregnancies. During this pregnancy, no fetal heart sounds were heard at eight months. Roentgenograms revealed a dead fetus in a transverse lie with signs of fetal hydrops. The patient has since had a normal full term baby with no trouble whatsoever.

of the soft parts. Unfortunately, we do not have postnatal roentgenograms for proper evaluation of this feature. When increased bone density is observed on prenatal roentgenograms, in the absence of characteristic changes of fetal hydrops, it should arouse suspicion of the presence of one of the milder forms of erythroblastosis fetalis. In

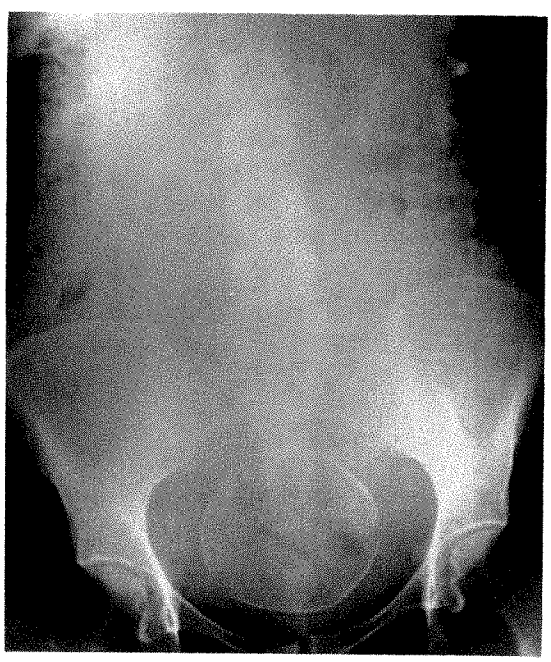


FIG. 7. Case 4. The mother was a thirty-six year old white female, para V. In 1949 a pregnancy resulted in hydramnios, fetal anomalies, and stillbirth. In 1951 the patient delivered an erythroblastotic baby at full term. Because of the mother's Rh negative blood, neonatal anemia and enlargement of the liver, replacement transfusion was done. This child is living and well at six years. In 1953 a full term living baby was delivered. Replacement transfusion was performed but the indications are not recorded in the history. In 1955 the patient delivered a three week premature baby. Neonatal death occurred as a result of Rh sensitization. This pregnancy was of seven months' duration. Hydramnios had been noted to progress during the past two months. The fetal movements were active. The patient experienced vaginal bleeding which lasted for four days and a roentgenogram revealed evidence of fetal hydrops. Five days later a hydropic infant was delivered which lived only twenty-five minutes (see Fig. 1, *A–C*).

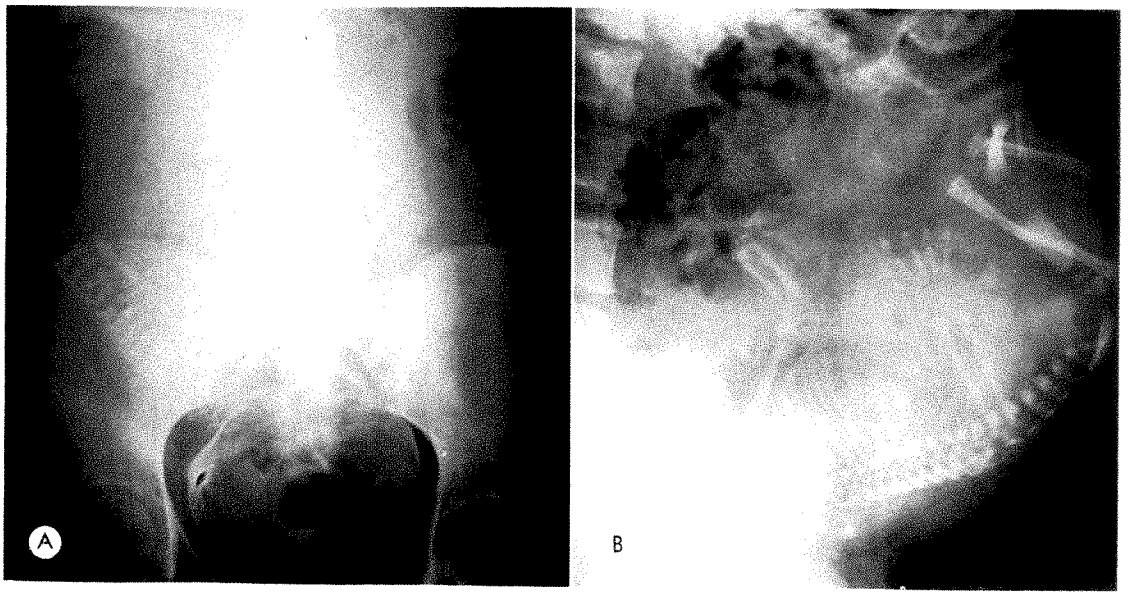


Fig. 8. Case 5. (*A* and *B*) The mother was a thirty-three year old white female, para III. In 1949 she delivered a normal child at term. In 1952 she also delivered an apparently normal child at term who, however, developed jaundice and facial edema the following day. The liver became enlarged and the general condition worsened. The child was found to have erythroblastosis due to a rare blood type, anti C (Fisher nomenclature), and developed cerebral palsy. The patient was admitted during this pregnancy at thirty-four weeks with hydramnios and painless bleeding. Roentgenograms made to determine the placental site or fetal anomaly were diagnostic of fetal hydrops. Cesarean section revealed a vigorous female child with severe fetal hydrops. The fetal weight was 2,730 gm., one quarter of which was estimated as due to edema. The liver and spleen were enlarged. Immediate transfusion with settled blood (Rh positive, Rh 1 homozygous) was given and 140 cc. of ascitic fluid was removed. A second exchange transfusion was performed when she was six and one-half hours old, a third on the third day and one when she was four days old. The child responded and is now well and flourishing.

postnatal studies this finding may be rewarding in making the diagnosis of questionable cases of fetal anemia or jaundice.

DISCUSSION

Erythroblastosis fetalis is a hemolytic clinico-pathologic syndrome of offspring, resulting from conception between an Rh negative woman and an Rh positive man. Fetal hydrops, the most severe form of this condition, is said to occur once in 2,000 deliveries. It is characterized by marked generalized edema of soft parts, associated with accumulation of fluid in the pericardial, pleural and peritoneal cavities, with enlargement of the heart, liver and spleen. The placenta is enlarged, thickened and edematous.

Due to advances in the care of premature infants and the spectacular results of prompt and repeated replacement blood transfusions, this condition is no longer a hopeless one. Early diagnosis and prompt therapy are essential. Roentgen studies are indicated whenever there is a clinical suspicion of hydramnios and in all pregnant women who are known to be Rh negative or who have previously had an erythroblastotic child.

The two best known diagnostic signs, the Buddha posture and the halo sign, have been unsatisfactory in the experience of many observers.

SUMMARY

Six cases of fetal hydrops correctly diagnosed by roentgenograms of the gravid abdomen are reported. The diagnostic features are based on both direct and indirect evidence of the pathologic changes:

1. Obliteration of the fetal fat line, due to subcutaneous edema.



Fig. 9. Case 6. The mother was a nineteen year old white female, para II. She was a known Rh negative who fifteen months previously delivered a full term fetus with fetal hydrops which lived only a few hours. A roentgenogram was made at thirty-four weeks to determine whether signs of erythroblastosis were present. There was no clinical suspicion of hydramnios, no toxemia, and no bleeding. The roentgenogram revealed findings typical of fetal hydrops and a cesarean section was performed. A markedly hydropic baby with an enlarged placenta was delivered and immediate replacement transfusion was given; however, the baby lived only two hours.

2. Displacement of the fetus an unusual distance from one side of the uterus, due to enlargement of the fetal abdomen and the placenta.

3. Elevation of the ribs, due to the enlarged heart and distended abdomen.

4. Peculiar posture of the arms and legs, due to soft tissue edema.

5. Intrauterine fetal death is a frequent finding.

In our experience the "halo" sign was not observed.

Chronologic review of our 6 cases gives us encouragement. The first 3 cases were dead at the time of the roentgen examination. The last 3 were delivered alive; one was salvaged, 2 died within short periods. It is our hope that this improvement is at least in part the result of the efforts of our clinico-roentgenologic teamwork and that our results can be projected to still more favorable levels in the future.

Department of Radiology
Pennsylvania Hospital
Philadelphia, Pennsylvania

REFERENCES

1. Bishop, P. A. Obstetrical roentgenology. In: Clinical Obstetrics. Edited by C. B. Lull and R. A. Kimbrough, Jr. J. B. Lippincott Company, Philadelphia, 1953, pp. 357–416.
2. Crane, A. R. Pathology of pregnancy and labor. In: Clinical Obstetrics. Edited by C. B. Lull and R. A. Kimbrough, Jr. J. B. Lippincott Company, Philadelphia, 1953, pp. 417–435.
3. Follis, R. H., Jr., Jackson, D., and Carnes, W. H. Skeletal changes associated with erythroblastosis fetalis. *J. Pediat.*, 1942, *21*, 80–92.
4. Hartley, J. B. Detection of foetal abnormalities. *Proc. Roy. Soc. Med.*, 1949, *42*, 301–310.
5. Hellman, L. M., and Irving, F. C. X-ray diagnosis of erythroblastosis. *Surg., Gynec. & Obst.*, 1938, *67*, 296–298.
6. Janus, W. L., and Dietz, M. W. Osseous changes in erythroblastosis fetalis. *Radiology*, 1949, *53*, 59–65.
7. Javert, C. T. Erythroblastosis neonatorum; obstetrical-pathological study of 47 cases. *Surg., Gynec. & Obst.*, 1942, *74*, 1–19.
8. Lee, H. F. Hemolytic diseases of new born. In: Clinical Obstetrics. Edited by C. B. Lull and R. A. Kimbrough, Jr. J. B. Lippincott Company, Philadelphia, 1953, pp. 643–648.
9. McClure Browne, J. C. Fallibility of radiological diagnosis of erythroblastosis foetalis. *J. Obst. & Gynaec. Brit. Emp.*, 1950, *57*, 71–72.
10. Pickles, M. M. Haemolytic Diseases of the New Born. Charles C Thomas, Publisher, Springfield, Ill., 1949.
11. Rittvo, M., Shauffer, I. A., and Krosnick, G. Clinical and roentgen manifestations of erythroblastosis fetalis. Am. J. Roentgenol. & Rad. Therapy, 1949, *61*, 291–301.
12. Samuel, E., and Cohen, J. Pre-natal radiological diagnosis of hydrops foetalis. *Brit. J. Radiol.*, 1950, *23*, 225–230.
13. Snow, W., and Powell, C. B. Roentgen visualization of placenta. Am. J. Roentgenol. & Rad. Therapy, 1934, *31*, 37–40.

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A NEW METHOD OF FETAL WEIGHT DETERMINATION*

By LEO STOCKLAND, M.D.,† *and* STANTON A. MARKS, M.D.‡
MILWAUKEE, WISCONSIN

THE existing methods of fetal weight determination have proved unsatisfactory at our Institution. It was felt that a more accurate method would have to be devised by which to ascertain the maturity of an infant before elective cesarean section or induction of labor is undertaken. The literature reveals approximately 5 per cent fetal mortality in elective cesarean sections due to prematurity.[1,3,6,9] This is a report of a new method of fetal weight determination which approaches satisfactory accuracy and is useful in cases when the obstetrician is not certain if the infant is mature enough to survive cesarean section or the induction of labor. Multiple measurements are used which when averaged together result in greater accuracy because of what we call the "equalizing factor."

EXISTING METHODS

The existing methods are based mostly on one measurement of the fetus. Following are the commonly used ones. The method of identifying the proximal tibial epiphysis which calcifies at the 39th or 40th week has been satisfactory if one can visualize it on the roentgenograms.[2] If the fetus is not of 39 or 40 weeks of gestation, the stage of maturity cannot be determined.

The distal femoral epiphysis calcifies at 35 to 40 weeks and again the same problem exists as in that of the proximal tibial epiphysis. Previous studies show that at 35 weeks the average fetus is 5 pounds and, therefore, by visualization of the distal femoral epiphysis, we cannot say unequivocally that a fetus is completely mature since the weight at this stage of gestation will vary from 5 pounds and up.[2]

The use of biparietal skull measurements allows us to consider only two-thirds of the cases examined due to lack of proper intrauterine position of the fetus. The literature reveals that with biparietal diameter measurements, two-thirds of the cases had an error of 1 pound greater or 1 pound less than the actual weight. Only infants weighing 5 pounds or more were used.[7] This does not take care of the "critical" range of 4 pounds to 6 pounds which we define as "that weight below which the fetus is obviously immature by clinical and/or roentgen methods and above which it is obviously mature by either or both methods."

The measurement by head volume reveals that this method will allow an error of 8 ounces or less in 36 per cent of the cases and an error of 16 ounces or less in 60 per cent of the cases.[4] However, all of the cases described in the literature in which this method was used were 6 pounds and 13 ounces or over, and, therefore, did not represent those of the "critical" weight group.

Other methods employed are those of measurement of fetal length, occipitofrontal diameter of the skull[10] and clinical estimation by measuring the uterus.[8]

INDICATIONS FOR ROENTGENOGRAPHIC EXAMINATION FOR FETAL WEIGHT

It is generally agreed that the indiscriminate use of roentgenography for determining fetal weight is to be avoided. Roentgenographic examinations, however, are indicated in cases where (1) elective cesarean sections are contemplated for the following reasons: (a) a repeat cesarean section is considered and the obstetrician is not certain of the degree of maturity of the

* From the Department of Radiology, Milwaukee County Hospital, Milwaukee, Wisconsin.
† Radiology resident.
‡ Attending radiologist.

fetus; (b) the mother is diabetic; (c) Rh problems exist; (d) cephalopelvic disproportion with a breech presentation is noted and, therefore, the patient cannot be given a trial of labor; (e) there is toxemia of pregnancy, and (2) the obstetrician may wish to induce labor and does not know the degree of maturity of the fetus.

RADIATION EXPOSURE

In order to reduce radiation to the maternal gonads, fetal gonads and to the entire body of the fetus to the least amount within practical limits, high speed screens with high speed film are recommended. A cone is used to collimate the roentgen-ray beam, and the beam is filtered with 2 mm. of aluminum which, in addition to approximately 0.5 mm. of aluminum inherent filtration, results in a total of 2.5 mm. of aluminum filtration. Webster and Merrill show that although the output varies among roentgenographic units, the anteroposterior projection results in an average of 250 mr to the maternal gonads and the lateral projection results in 280 mr to the gonads. The average total dose for the examination is 530 mr to the maternal gonads. There have been no studies of radiation received by the fetus itself and, therefore, it must be assumed that the dosage is slightly above that received by the maternal gonads in the anteroposterior projections, since the fetus is closer to the roentgen-ray tube. In the lateral projection there is probably very little difference. These figures are obtained with a 200 milliampere unit. A dose of 500 to 550 mr to the fetus and maternal gonads should not contraindicate an examination which may increase the survival rate of infants delivered by cesarean section.

TECHNIQUE

The success of our method depends upon satisfactory roentgenograms so that the landmarks may be accurately identified. Two roentgenograms are taken, one in the anteroposterior and one in the lateral projection. Wire abdominal markers are taped across the anterior portion of the abdomen for the lateral view and across the two lateral margins of the abdomen for the anteroposterior view. Solder wire has been used since it is very pliable and is radiopaque. This delineates the abdominal wall and, therefore, assists in obtaining measurements of the uterus with a greater degree of reliability. A constant tube film distance of 40 inches is used. In the graphs which are constructed, the average distortion produced by a 40 inch target film distance is considered, and, therefore, no correction factor need be used.

It is possible that logEtronic reproduction, by clearly delineating the fetal points of measurement, may increase the accuracy of the procedure; this method will be used in some of the future cases.

The radiologist should position the patient and check the roentgenograms before the patient is dismissed to ensure that they are adequate for measurement. The anteroposterior roentgenogram is taken first so that a determination of a cephalic or breech presentation may be made. If a breech presentation is present, the lateral roentgenogram should be taken with the fetal head closest to the film.

CONSTRUCTION OF GRAPHS

The results obtained in the examination of 50 patients were used to construct graphs relating the fetal weight to various measurements. Scatter graphs were made using known weights versus known measurements, where the fetus was delivered within a day or so following the taking of roentgenograms. When the patient did not deliver for a significant time interval after roentgenography, the weight of the fetus at the time of roentgenography was computed from a gestation versus weight table, which was constructed from Streeter's tables of 704 specimens.[5]

PROCEDURE

The measurements which are usually made are illustrated in Figure 1, *A* and *B*.

Step 1. Skull Measurement (Fig. 2). This

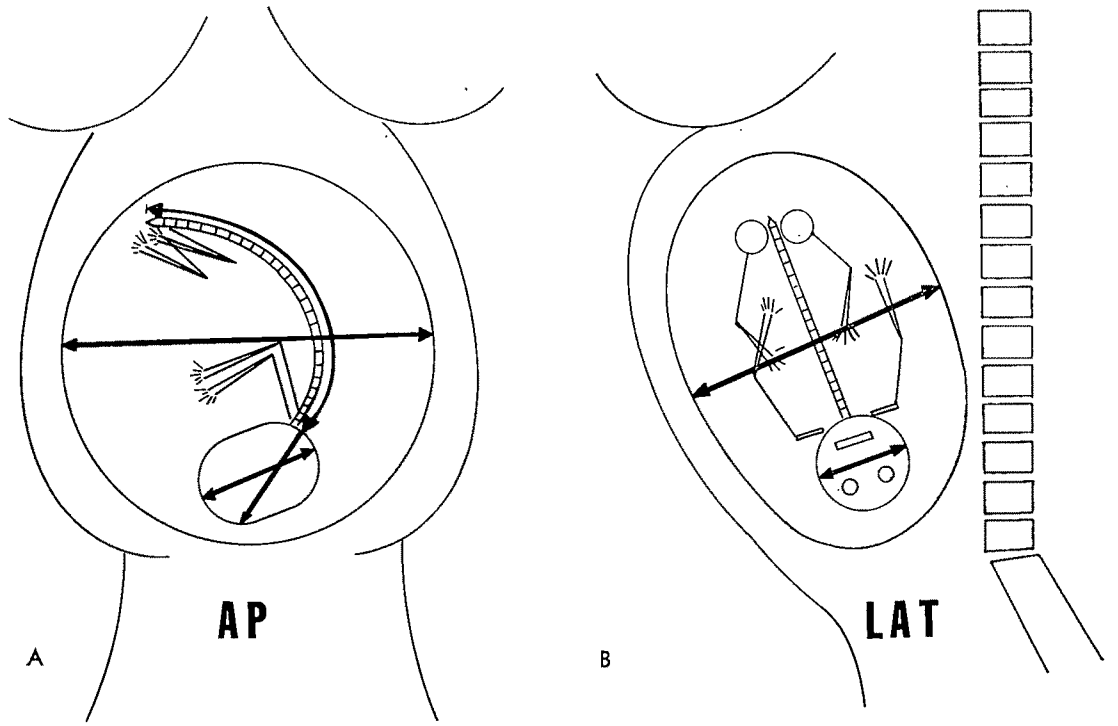


Fig. 1. (*A* and *B*) Schematic sketch showing measurements to be taken.

is found by (1) measuring the biparietal diameter of the skull; (2) measuring the occipitofrontal diameter of the skull, and (3) adding these two measurements together.

Generally, it is possible to obtain the biparietal measurement from the lateral roentgenogram and the occipitofrontal measurement from the anteroposterior roentgenogram. If only the biparietal diameter can be determined from both the anteroposterior and lateral roentgenogram, we have found that by adding 20 per cent to the biparietal diameter, we can arrive at the average occipitofrontal diameter. Conversely, if only the occipitofrontal measurement can be obtained, the biparietal measurement can be determined by subtracting 20 per cent from this measurement based on an average cephalic index of 8:10. This is the ratio of the broad diameter to the long diameter in the elliptically shaped skull and thus the sum of the biparietal and the occipitofrontal diameter cannot be in error by more than approximately 6 per cent. If the head is oblique in both projections, these two measurements are still added together.

(4) The fetal weight is taken from the skull versus weight graph.

Step II. Length Measurement (Fig. 3). This is found by (1) placing one point of a plastic flexible ruler at the coccyx and

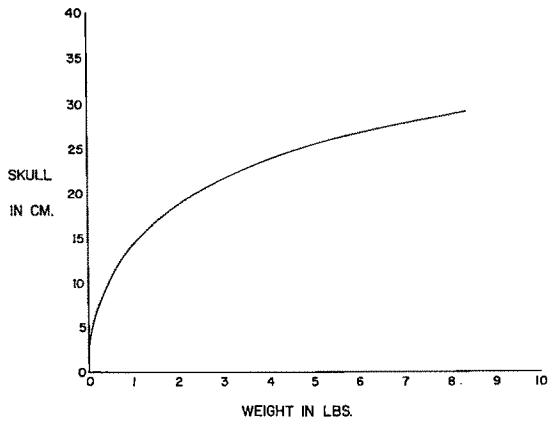


Fig. 2. Graph showing fetal weight versus skull measurements.

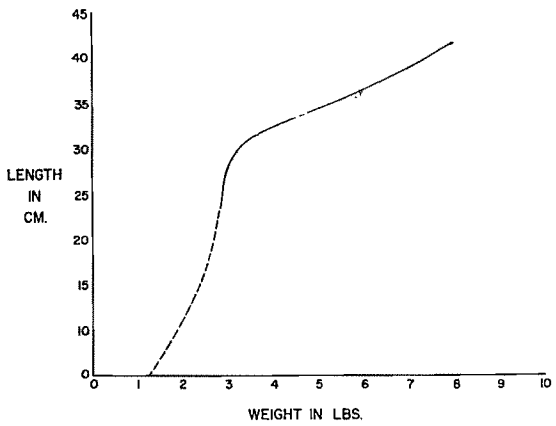


FIG. 3. Graph showing fetal weight versus length measurements.

measuring to the odontoid process along the contour of the spine; (2) drawing a line from this point to a point on the outer table of the skull which is at the greatest distance from the odontoid process; and (3) adding these two measurements together and thus obtaining the necessary figures for the total length versus weight graph. Here again, the graph was designed using the scatter graph method of known length versus known weight.

Step III. Uterus Measurement (Fig. 4). This is found by (1) measuring the greatest width of the uterus from the anteroposterior roentgenogram and the greatest width of the uterus from the lateral roentgenogram; and (2) adding these two measurements and then plotting them on the uterus measurements versus weight graph.

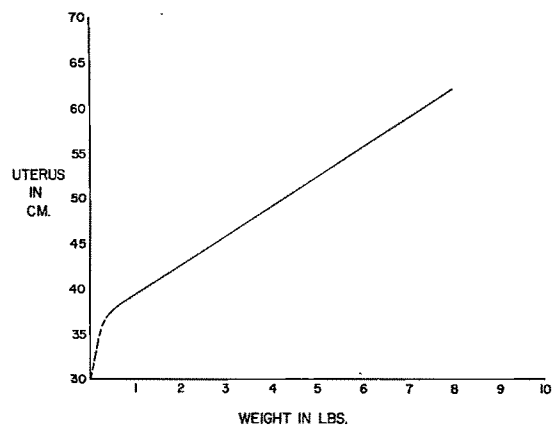


FIG. 4. Graph showing fetal weight versus uterus measurements.

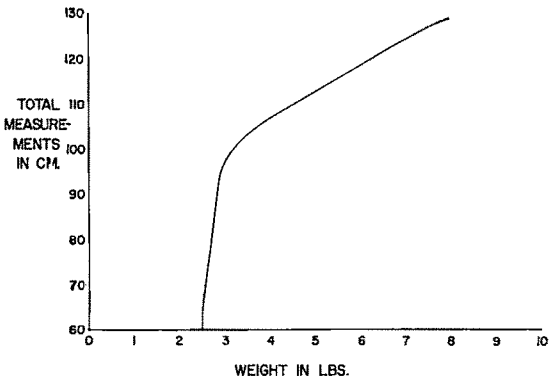


FIG. 5. Graph showing fetal weight versus total measurements.

Step IV. Totaling of Measurements (Fig. 5). This is done by (1) adding the measurements of the skull, length, and uterus; and (2) by plotting the sum on the total measurements versus weight graph.

Step V. Averaging. The weights obtained from the skull, length, uterus, and sum graphs are then totaled, and the average is found. This is the average or estimated weight of the fetus on the day of roentgenography.

It will be noted that the weights computed separately from the skull, length and uterus measurements may vary but the equalizing factor of the average weight results in the true weight.

Step VI. Gestation Determination (Fig. 6). This is found (1) after the estimated weight of the fetus has been determined. The expected weight increase may be obtained by using the gestation versus weight graph; then (2) the weight of the fetus at the time of roentgenography is recorded on the gestation weight graph. From the graph the number of weeks necessary for the desired increase in the weight of the fetus can be read.

UNUSUAL FETAL POSITIONS

Breech Presentation. Measurements are made in the same manner as in cases of cephalic presentation. The only change is

that the lateral roentgenogram is taken so that the head of the fetus is closest to the film. This can easily be determined after viewing the anteroposterior roentgenogram.

Transverse Presentation. After viewing the anteroposterior roentgenogram, the lateral roentgenogram is taken so that the head of the fetus is closest to the cassette. The skull and length measurements are determined in the same manner as in case of cephalic presentation. The measurement of the uterus from the anteroposterior roentgenogram is obtained in a cephalocaudal instead of the transverse dimension. On the lateral roentgenogram the measurement is taken through the greatest width of the uterus. All of the computations are then made in the same manner as for cephalic presentation.

Twin Pregnancy. Measurements are taken of only one of the intrauterine fetuses since twins are approximately of the same weight. The skull and length measurements are determined as in a single fetus. The uterus measurement is taken through the greatest width on both the anteroposterior and lateral roentgenogram. Seventy-five per cent of the total of the two measurements is used in computation since only the weight of one of the intrauterine fetuses is to be determined.

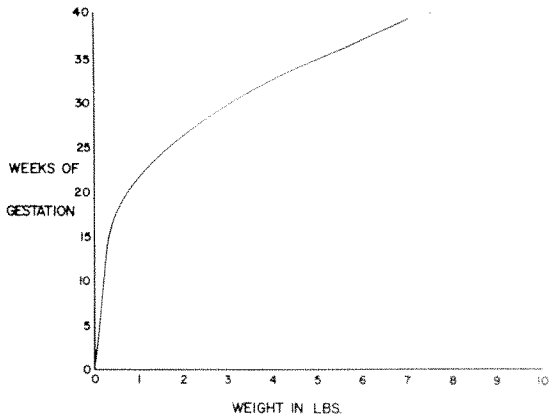


FIG. 6. Graph showing fetal weight versus weeks of gestation.

REPORT OF CASES

CASE 1. M.J.A. This twenty-two year old pregnant Negro female (Fig. 7, *A* and *B*), who had a previous cesarean section, entered Milwaukee County Hospital with an unreliable

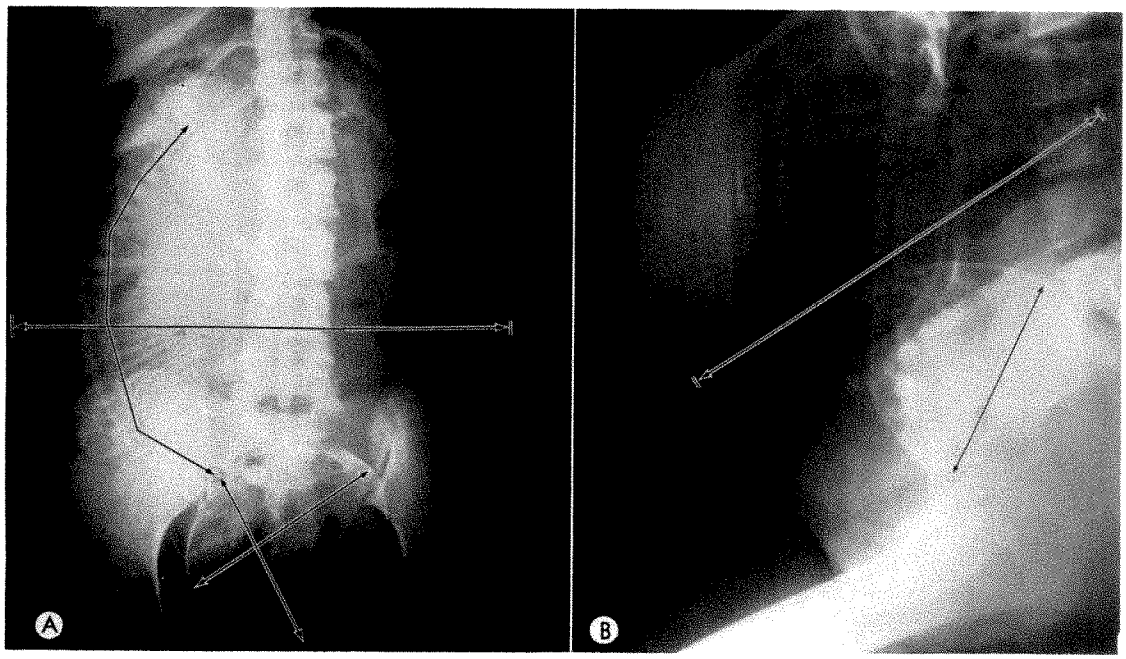


FIG. 7. (*A* and *B*) Case 1.

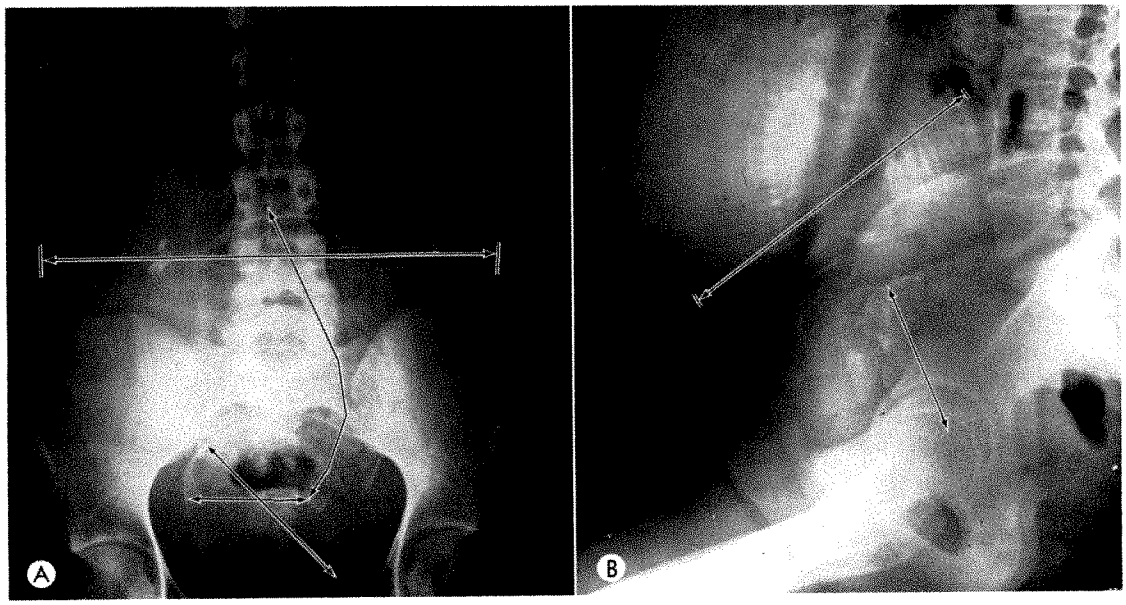


FIG. 8. (*A* and *B*) Case II.

menstrual history. The following statistical data were determined: skull—24.5 cm., 5 pounds; length—37.5 cm., 6 pounds 8 ounces; uterus—52.5 cm., 5 pounds; total—114.5 cm., 5 pounds 9 ounces; average weight—5 pounds 8 ounces; duration of gestation—36 weeks; time interval between roentgenography and birth—2 weeks; actual birth weight—6 pounds 3 ounces; estimated birth weight—6 pounds 8 ounces; error—5/101 = +5 per cent.

Comment. The average or estimated weight at the time of roentgenography was 5 pounds 8 ounces, which was approximately at 36 weeks of gestation. We suggested that an interval of two weeks should elapse before a cesarean section be done since our weight versus gestation graph revealed that at that time the fetus would weigh 6 pounds 8 ounces. This was done, and the actual birth weight was 6 pounds 3 ounces. By using this method premature birth was prevented.

CASE II. T.R. This twenty-eight year old white female (Fig. 8, *A* and *B*) entered the hospital for reasons unrelated to her pregnancy. Here again, the actual menstrual history was completely unreliable and roentgenograms were taken for future reference. The following statistical data were determined: skull—19.4 cm., 2 pounds 6 ounces; length—28 cm., 3 pounds; uterus—41.5 cm., 1 pound 12 ounces; total—88.9 cm., 2 pounds 9 ounces; average weight—2 pounds 7 ounces; duration of gestation—28 weeks; time interval between roentgenography and birth—11½ weeks; actual birth weight—7 pounds 6½ ounces; estimated birth weight—7 pounds 8 ounces; error—1.5/118.5 = +1 per cent.

Comment. This patient had a normal uneventful delivery. Nevertheless, by our method we were able to predict within 1 per cent the birth weight of the baby.

CASE III. This twenty-eight year old white female (Fig. 9, *A* and *B*) with an unreliable menstrual history entered the hospital for evaluation. The following statistical data were determined: skull—24.8 cm., 5 pounds 2 ounces; length—39 cm., 7 pounds; uterus—61.2 cm., 7 pounds 3 ounces; total—125 cm., 7 pounds 10 ounces; average weight—6 pounds 12 ounces; duration of gestation—38½ weeks; time interval between roentgenography and birth date—11 days; actual birth weight—7 pounds 6 ounces; estimated birth weight—7 pounds 8 ounces; error—2/118 = 2 per cent.

RESULTS

The degree of accuracy in a series of 179 cases is graphically illustrated in Figure 10.

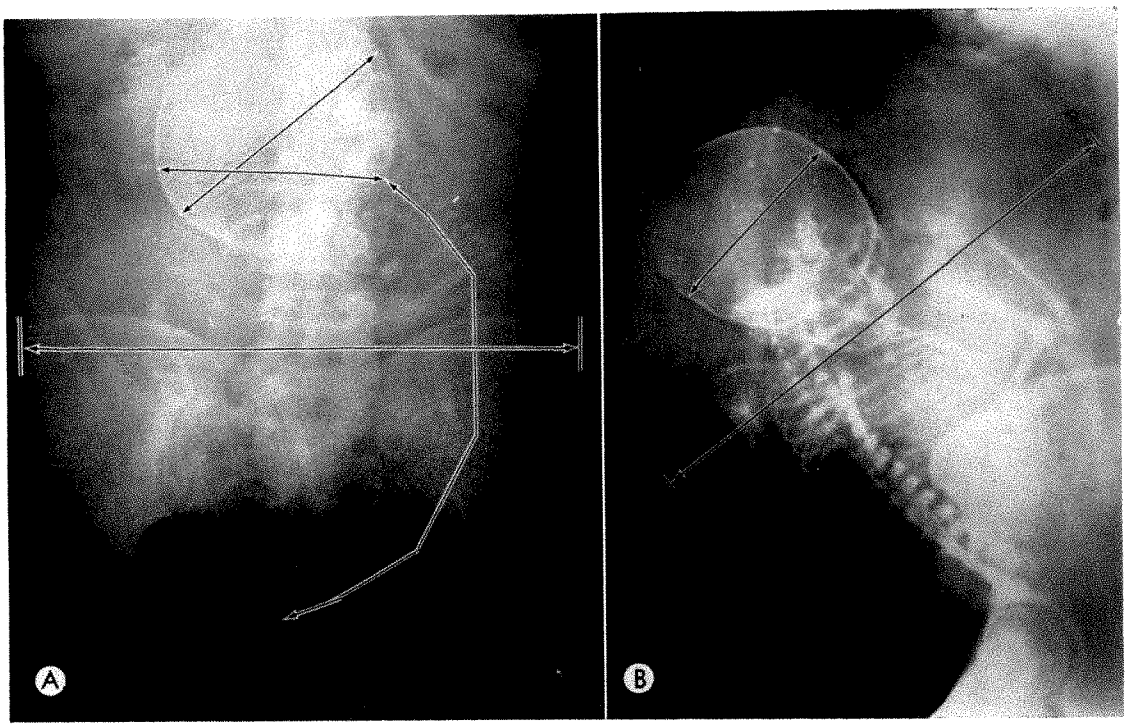


FIG. 9. (*A* and *B*) Case III.

SUMMARY

1. A report of a new method of fetal weight determination has been presented.
2. It is believed that its degree of accuracy is superior to that of other existing methods.
3. The ability to project the weight at birth is possible with this method.

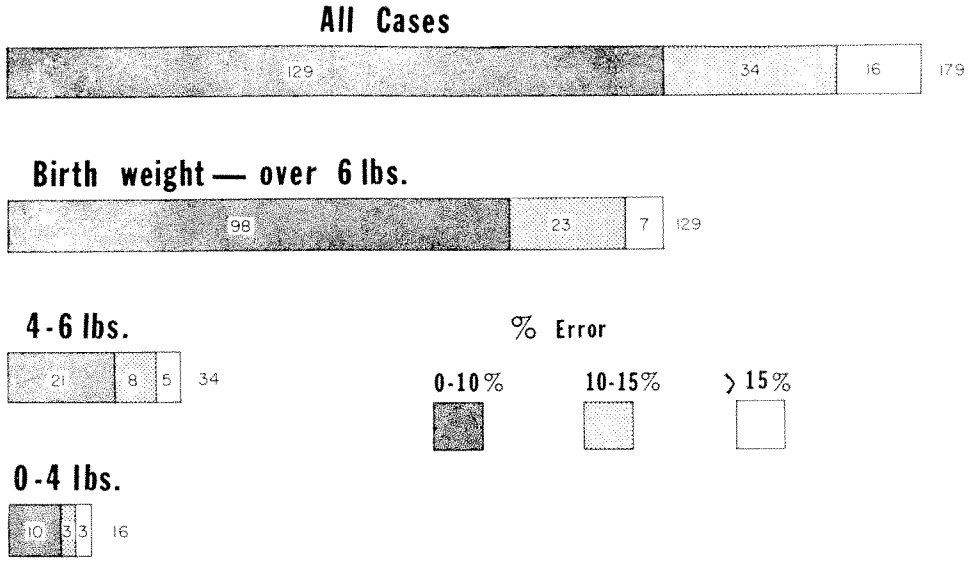


FIG. 10. Diagram showing the birth weight error.

4. Radiation exposure of the maternal gonads and fetus should not contraindicate an examination which may save the lives of 5 per cent of babies who are delivered by elective cesarean section.

Stanton A. Marks, M.D.
161 West Wisconsin Avenue
Milwaukee 3, Wisconsin

The authors wish to thank Dr. John R. Amberg, Chief of Radiology, John A. Lind and Calvin Williams, radiology residents, for their help in making this report possible.

REFERENCES

1. BUBALO, J. M. Repeat cesarean sections and perinatal mortality. *Obst. & Gynec. Survey*, 1957, *12*, 862–864.
2. CHRISTIE, A., MARTIN, M., WILLIAMS, E., HUDSON, G., and LASSIER, J. Estimation of fetal maturity by roentgen studies of osseous development. *Am. J. Obst. & Gynec.*, 1950, *60*. 133–139.
3. DIDDLE, A. W., GIBBS, V., and LAMBETH, S. Fetal mortality and prematurity with repeat abdominal delivery. *Am. J. Obst. & Gynec.*, 1959, *77*, 719–725.
4. DONALDSON, S. W., and CHENEY, W. Prenatal estimation of birth weight by pelvicephalometry. *Radiology*, 1948, *50*, 666–667.
5. EASTMAN, N. J. Williams Obstetrics. Tenth edition. Appleton-Century-Crofts, 1950, p. 168.
6. GROSSMAN, M. Fetal mortality in cesarean section. *J.A.M.A.*, 1956, *162*, 1289–1293.
7. JACOBS, J. B. Value of lateral pelvic roentgenogram as index of fetal maturity and type of maternal pelvis. *Am. J. Obst. & Gynec.*, 1953, *65*, 897–908.
8. JOHNSON, R. W., and TOSHACH, C. E. Estimation of fetal weight using longitudinal mensuration. *Am. J. Obst. & Gynec.*, 1954, *68*, 891–896.
9. JOHNSTON, R. A., and MORGAN, J. R. Analysis of 641 repeat cesarean sections. *South. M. J.*, 1957, *50*, 764–767.
10. SHANKS, S. C., and KERLEY, P. Textbook of X-Ray Diagnosis. Vol. III. W. B. Saunders Company, Philadelphia, 1958, p. 583.

PREMATURE UNILATERAL SYNOSTOSIS OF THE CORONAL SUTURE*

By M. H. NATHAN, M.D., V. P. COLLINS, M.D., *and* L. C. COLLINS, M.D.
HOUSTON, TEXAS

PREMATURE, unilateral, coronal synostosis (hereinafter referred to as unilateral coronostenosis) is a congenital abnormality of the skull with a distinctive combination of roentgenographic features. Failure to distinguish this entity from certain other diseases that affect the configuration of the skull may lead to unnecessary diagnostic procedures and to delayed or inappropriate treatment. McLaurin and Matson[12] advocate the earliest possible treatment for the prevention of exophthalmos, visual loss, cosmetic deformities and perhaps brain damage.

Since the most important findings necessary for early diagnosis are the roentgenographic signs, the radiologist has the responsibility of establishing or confirming the diagnosis. Because it is uncommon, this condition is likely to present a problem in roentgenologic interpretation. Case presentations and discussion of this form of craniostenosis, with emphasis on clinical and roentgenologic diagnosis, should make immediate recognition possible.

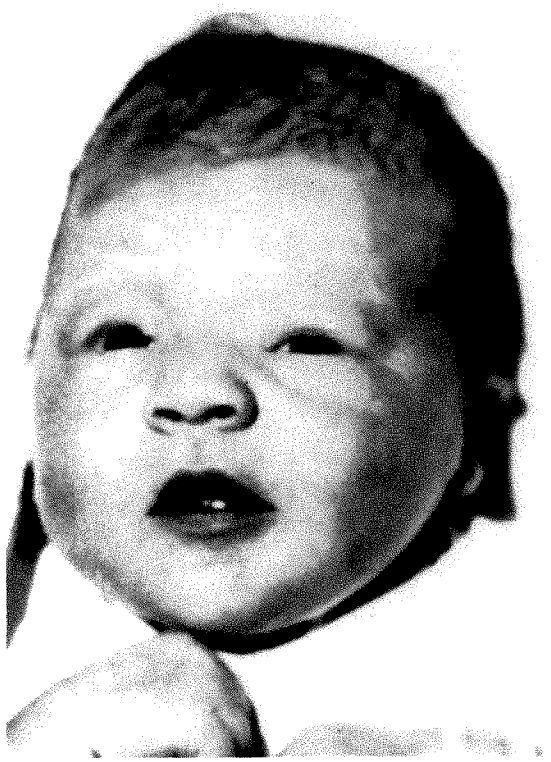


FIG. 1. Case 1. A one month old white female with right coronal synostosis. There is flattening of the right frontoparietal region, right sided exophthalmos, and minimal facial asymmetry.

REPORT OF CASES

CASE 1. T.C., a white female infant, was brought back to the physician who delivered her for a routine examination at the age of one month. At this time, the doctor noticed a slight exophthalmos of the right eye, flattening of the right frontal area of the skull, and minimal asymmetry of the face (Fig. 1). There were no other positive findings; development was normal and the neurologic examination revealed no abnormalities.

Roentgenograms of the skull demonstrated narrowing of the right coronal suture with fusion of its inferior portion (Fig. 2, *A* and *B*); this was more readily apparent on the roentgenograms made for the optic foramen (Fig. 3, *A* and *B*) which also showed narrowing of the sphenofrontal, sphenoparietal, squamosal, and sphenosquamosal sutures. The Water's projection (Fig. 4, *A* and *B*) revealed an elliptical shape to the enlarged right orbit, elevation of the right lesser sphenoid wing, slight flattening of the right parietal area, and slanting of the nasal septum with its cephalad end pointing to this same side. In the Towne view (Fig. 5, *A* and *B*), flattening of the parietal bone was more evident posteriorly and was associated with displacement of the posterior sagittal suture and lambda to the right. In addition, there was depression of the right petrous ridge and deepen-

* From the Department of Radiology, Baylor University College of Medicine, Texas Medical Center, Houston, Texas.

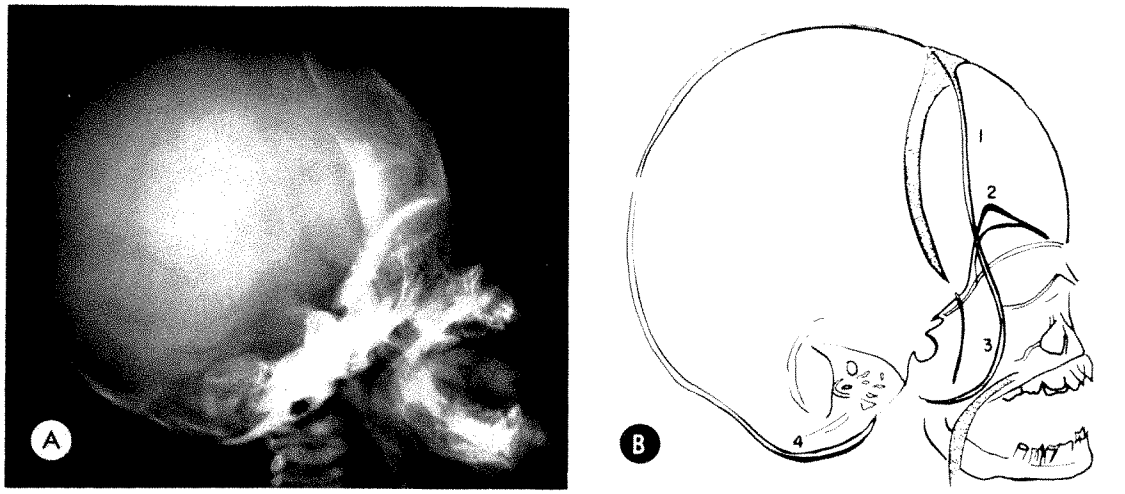


FIG. 2. Case 1. (*A* and *B*) Roentgenogram and its tracing showing narrowed right coronal suture (1); elevated right orbit (2); enlarged middle (3) and posterior (4) fossae. The right anterior fossa is shortened.

ing of the right middle and posterior fossa. Shortening of the anterior fossa with forward displacement of the anterior margin of the middle fossa was seen on the lateral roentgenogram (Fig. 2, *A* and *B*). Although the skull appeared to have an increased height, measurements were within the upper limits of normal.[5] There was no asymmetry in thickness of the calvarium and no increase in convolutional markings.

Corrective surgery was performed for right coronostenosis in the following manner: a small-nosed rongeur was used to make a false suture behind the right coronal suture. Polyethylene sheeting was placed between the cut edges and held in place by three sutures. The postsurgical course was uneventful. The infant was lost to follow-up two months later before postsurgical roentgenograms were made.

CASE II. M.M.L., a two and one-half year old female, had been in an automobile accident on January 5, 1960. She was never unconscious and did not exhibit any signs or symptoms indicative of brain injury, but the parents noted that the head was abnormal in appearance. The neurosurgeon to whom the child was referred reported discoloration around the right eye; a large and asymmetric head; high pitched percussion note; head measurements 2.5 cm. larger than normal for a child of this age; an unsatisfactory funduscopic examination, but there appeared to be some hemorrhage; equal and active deep reflexes; and normal muscle strengths in all extremities. It was not until later when he was asked to look carefully for this specific sign that the physician noted slight facial asymmetry. The child's development had been normal.

The skull roentgenograms revealed flattening and thinning of the parietal region, elliptical orbit with decreased depth, elevation of the lesser sphenoid wing, shortened anterior fossa, forward displacement of the greater sphenoid wing, enlargement of the middle and posterior fossae, and depression of the petrous ridge. All of these findings were asymmetrically located on the right (Fig. 6, 7 and 8). Mild obliquity of the nasal septum was also noted with its superior end pointing to the right and the posterior sagittal suture and lambda displaced considerably in the same direction. The length and breadth of the skull measured within limits of normal, but the height was increased. These roentgenograms were made elsewhere and the only lateral views available were some made at the time of pneumoencephalography. The coronal sutures were very poorly demonstrated and could not be evaluated. The radiologist considered tumor or hydrocephalus as well as unilateral, partial, and premature closure of the right coronal suture in the differential diagnosis. Pneumoencephalography was interpreted as normal.

Surgical correction for unilateral coronostenosis was performed by the neurosurgeon in the same manner as in the preceding case.

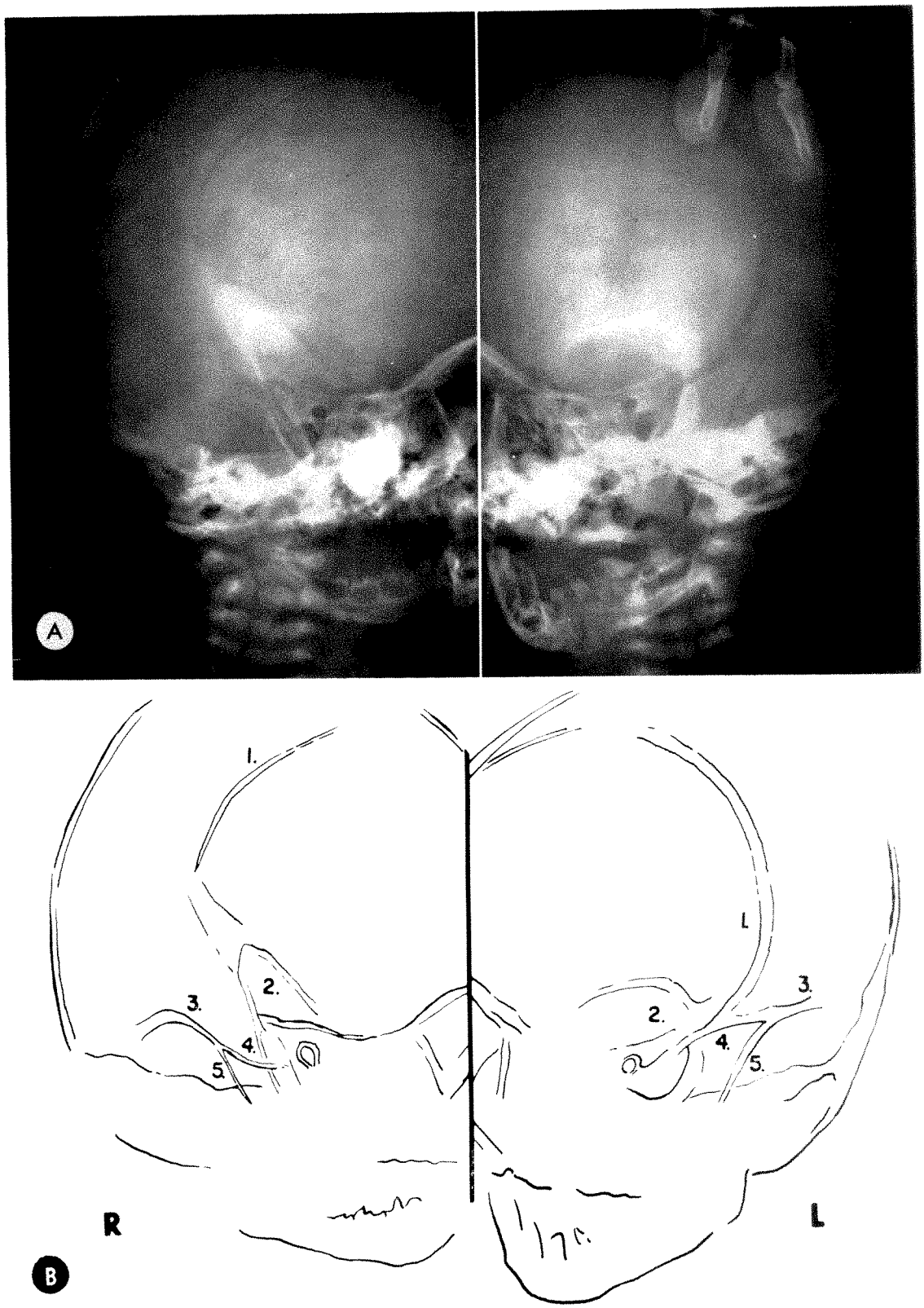


FIG. 3. Case 1. (*A* and *B*) The optic foramina are normal. The entire right coronal suture (1) on the right side is narrowed and synostosis is taking place in its inferior portion. There is also narrowing of the right squamosal (3), sphenoparietal and sphenofrontal (4), and sphenotemporal (5) sutures. The elliptical configuration of the right orbit (2) is well shown.

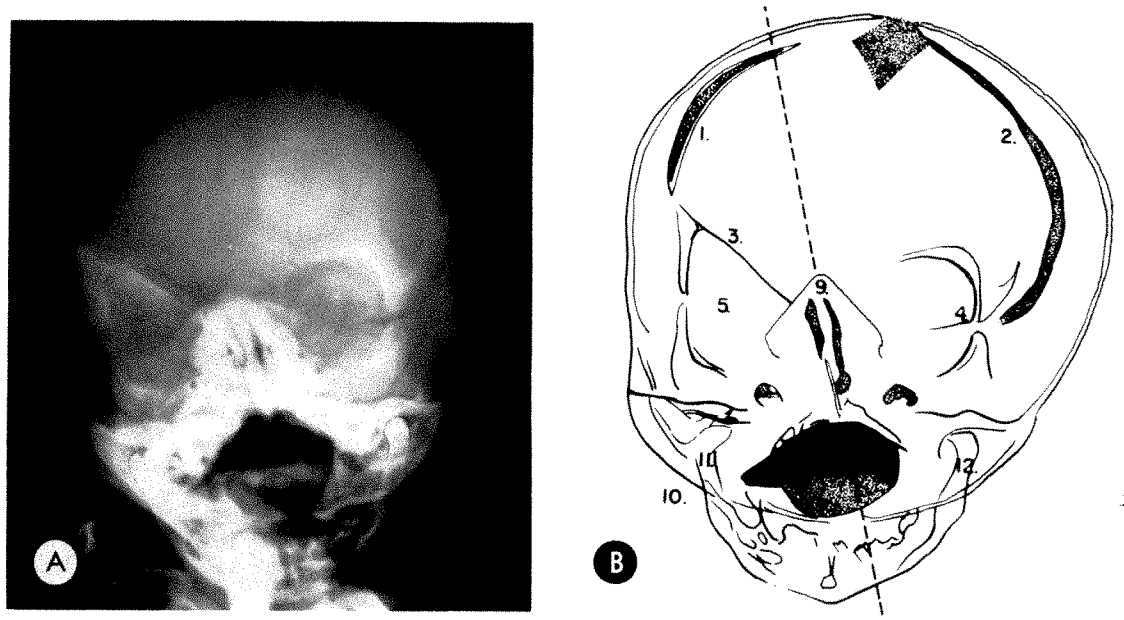


FIG. 4. Case 1. (*A* and *B*) Water's view showing elevation of the right sphenoid wing and sphenoid ridge (3), flattening of the right parietal area and obliquity of the nasal septum (9) with its superior end pointing to the side of the pathology. Right and left coronal suture (1 and 2); sphenoid wings (3 and 4); enlarged, elliptical right orbit (5); depressed right mandibular condyle (retouched) (11); and left mandibular condyle (12).

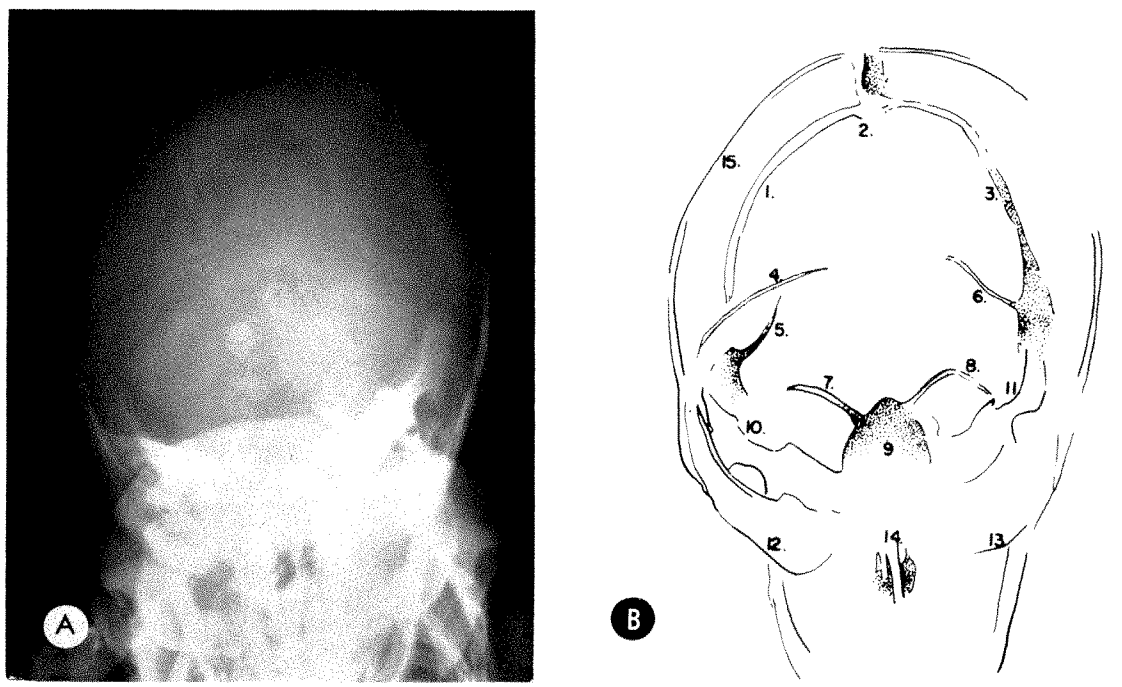


FIG. 5. Case 1. (*A* and *B*) Flattening of the right parietal region (15) is shown best in this Towne-Chamberlain projection as is the depression of the petrous ridge (10) on the same side. Lambdoidal sutures (1 and 3); lambda (2) displaced to right, mendosal sutures (5 and 6); narrowed right coronal suture (4); synchondrosis between ex-occipital and supra-occipital (7 and 8); depressed right petrous (10) and normal left petrous (11) ridges; depressed right middle (12) and normal left middle (13) fossae; and an oblique nasal septum (14) are also seen.

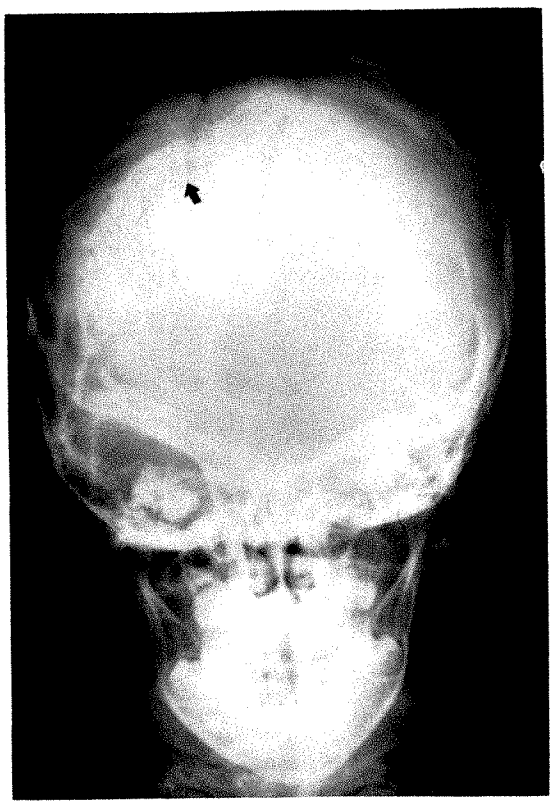


Fig. 6. Case II. Posteroanterior roentgenogram showing enlarged elliptical orbit, elevated sphenoid wings and deepening of the middle fossa, all on the right. There is also slight obliquity of the nasal septum and displacement of lambda to the right (arrow). Depression of the right middle fossa has resulted in the lower position of the right mandibular condyle (retouched).

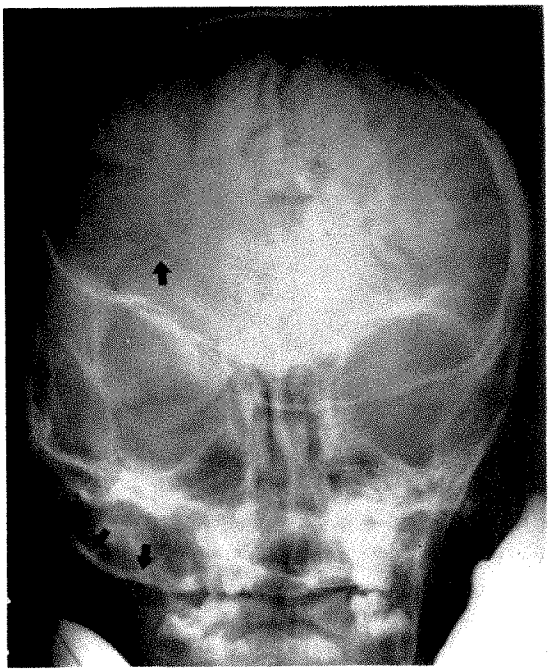


Fig. 7. Case II. Roentgenogram of the sphenoid area showing the same findings as described in Figure 6. In addition, the double arrows point to the enlarged right posterior fossa. The single arrow points to lambda. This roentgenogram was made during pneumoencephalography.

Case III. S.B., a seven and one-half month old white female, was brought to the clinic because of irritability, particularly in the last three months, and also because of flattening of the right frontal region which has not been progressive. The mother had many roentgenograms and several operations for intestinal obstruction during the gestation period. The child was born prematurely but her development has been normal.

Physical examination revealed an irritable baby with flattening of the right frontal bone, elevation of the right orbital rim, elevation of the right eyebrow and slight facial asymmetry. There was also flattening of the right occipital region of moderate degree. The head size measured within normal limits. The motor reflex and sensory testing were normal. The ocular

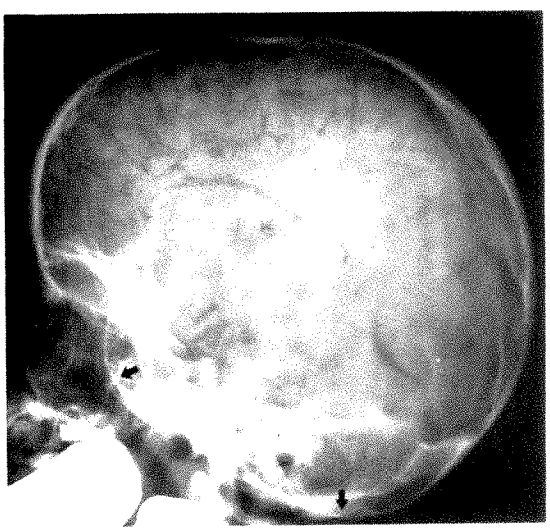


Fig. 8. Case II. Roentgenogram showing ventral bulging and apparent enlargement of the right middle fossa and enlargement of the right posterior fossa (arrows). This roentgenogram was made during pneumoencephalography.

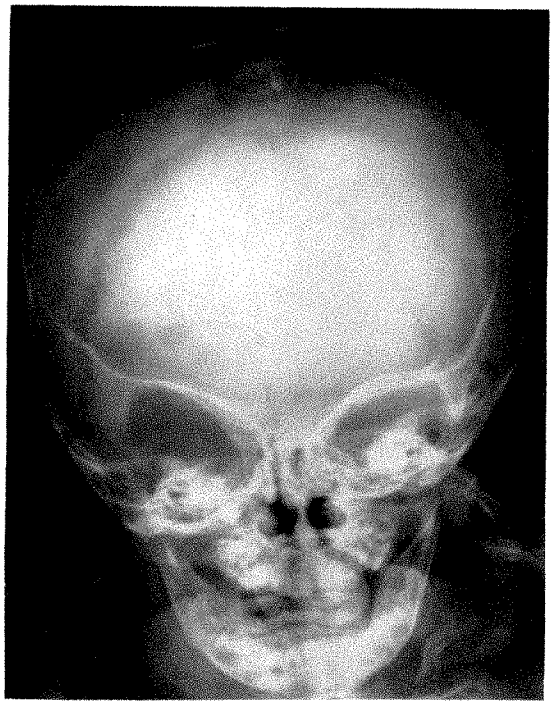


FIG. 9. Case III. The obliquity of the nasal septum, displacement of lambda, elevated right sphenoid wing, and the elliptical right orbit are prominent findings. Asymmetry of the skull, depression of the right middle fossa, mandibular condyle, petrous ridge and posterior fossa are also shown.

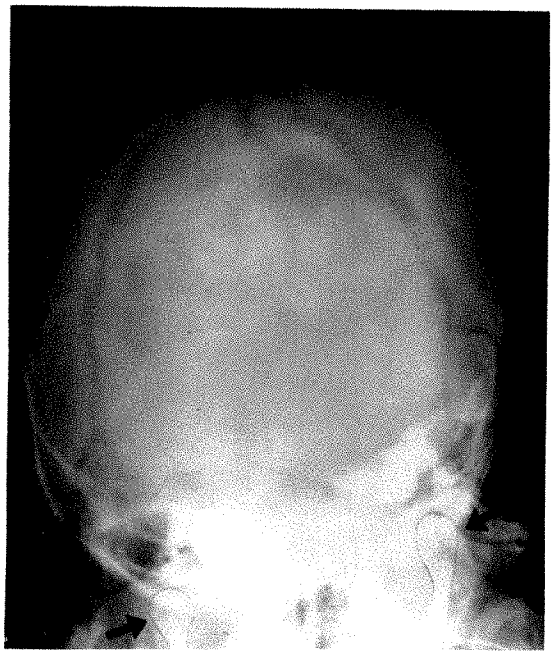


FIG. 10. Case III. Asymmetry with flattening of the right parietal bone, depression of the right middle and posterior fossae and the right mandibular condyle (retouched) are shown best on this roentgenogram. The lambda is displaced to the right.

movements were of full range and the optic fundi were normal. The vision and hearing seemed to be normal. The electroencephalogram was normal.

Roentgenograms showed right, unilateral coronostenosis with associated findings as demonstrated in Figures 9, 10 and 11 and as indicated in Table I.

A channel, 1.5 cm. wide, was made along the right coronal suture and extended down below the squamosal suture. No removal of orbital roof or lateral orbital wall was carried out. Polyethylene film was placed over the edges of this bone and heavy silk sutures were tied about the film through holes punched in the adjacent bone.

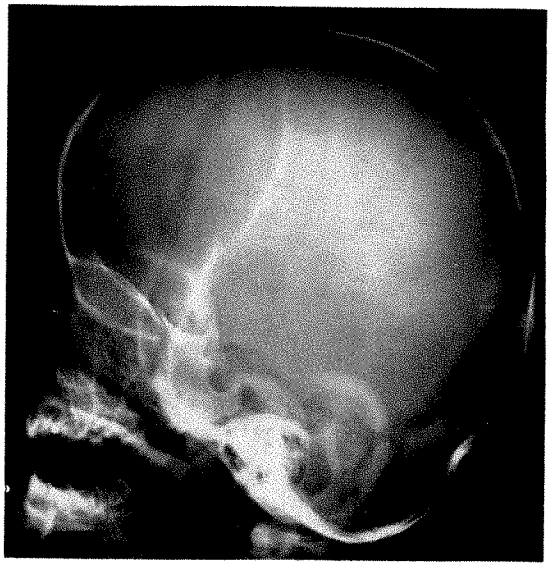


FIG. 11. Case III. Depression of the right posterior fossa appears to be the most prominent finding on the lateral roentgenogram. The deepening of the right middle fossa is less apparent and no bulging of its anterior margin is demonstrated.

GENERAL CLINICAL DISCUSSION

Unilateral coronostenosis is a congenital disease of unknown etiology with a familial tendency. The signs of synostosis are usually present at birth. Immediate postnatal recognition of the skull abnormality by the

TABLE I

FREQUENCY OF THE MOST IMPORTANT DIAGNOSTIC ROENTGENOGRAPHIC SIGNS OF UNILATERAL CORONOSTENOSIS IN 11 CASES (8 previously reported)

Roentgenographic Signs	Case I T.C.	Case II M.M.L.	Case III S.B.	Case IV[7]	Case V[19]	Case VI[12]	Case VII[16]	Case VIII[4]	Case IX[13]	Case X[9]	Case XI[18]
Orbit—Elliptical, Elevated and/or Enlarged	+	+	+	+	+	+	+	+	+	+	o
Lesser Sphenoid Wing—Elevated	+	+	+	+	+	+	+	+	+	+	o
Parietal Bone—Flattened	+	+	+	+	+	+	+	+	+	+	+
Parietal Bone—Thinned	−	+	−	−	−	−	−	−	+	+	−
Anterior Fossa—Shortened	+	+	o	+	o	o	o	o	o	+	o
Middle Fossa—Enlarged	+	+	+	o	o	o	o	o	o	o	o
Posterior Fossa—Deepened	+	+	+	+	+	+	+	+	+	o	+
Petrous Ridge—Depressed	+	−	+	+	+	−	+	+	+	+	+
Temporomandibular Fossa—Depressed	+	+	+	+	o	o	+	o	o	o	+
Nasal Septum—Obliqued	+	+	+	+	−(?)	+	+	o	+	o	+
Lambda—Deviated	+	+	+	+	+	o	+	−	o	−	o
Clinical Facial Asymmetry	+	+	+	+	+	+	o	o	o	o	o

Note: In most of the previously reported cases, only a single posteroanterior roentgenogram was available for study.
+ indicates that the sign was present.
− indicates that the sign was absent.
o indicates that the sign was not visualized well enough to evaluate.

clinician may be hampered by edema of the scalp and moulding of the skull secondary to the birth process but, as these effects subside, the asymmetry of the skull becomes more apparent. Consistently present is flattening of the frontal region; exophthalmos and facial asymmetry may be apparent clinically.

Failure to realize that unilateral exophthalmos may be associated with this disease can lead to confusion and erroneous diagnosis. Other causes of exophthalmos in this age group include brain tumor, subdural hematoma, arteriovenous aneurysm of the brain or orbit, orbital neoplasm, congenital cyst and hemangioma of the orbit, orbital hemorrhage, and defective development of the sphenoid bone. Flattening of the ipsilateral frontoparietal region would not be expected in these, however,

and exophthalmos associated with flattening therefore becomes a significant clinical sign for unilateral coronostenosis. The exophthalmos which occurs in defective development of the sphenoid bone is usually found to pulsate.[14] Pulsating exophthalmos also occurs in arteriovenous aneurysm of the brain, orbit and cavernous sinus, and a bruit is often associated in these cases.[17]

Asymmetric eye signs in unilateral coronostenosis are due to the deformity of the orbit on the involved side[11] and to premature fusion of the orbital bones.[12] Hyperopia results from shortening of the orbit and is sometimes associated with astigmatism; divergent strabismus is common[11] and optic atrophy may occur with blindness[13] due to bony pressure on the optic nerve.[19]

Asymmetric flattening of the skull may occur in other entities and is most often due to longstanding postural pressure by chronic resting of the head in one position during infancy; this is usually seen in defective infants who move their heads little and in infants with soft skulls.[4] The appearance of these infants may at times cause the clinician to think of plagiocephaly, but the history, clinical observations and roentgenographic findings should help to establish the correct diagnosis. Asymmetry of the skull also may occur with scoliosis and torticollis, which have been referred to as scoliosis capitis,[9] but this occurs later and should present no difficulty in differentiation. The asymmetry seen in hemiatrophy of the brain occurs later in infancy or childhood than that due to a craniostenosis, and the degree of asymmetry is less obvious. Nevertheless, where exophthalmos is absent, cerebral hemiatrophy must be a serious consideration. Subdural hematoma and brain tumor sometimes cause skull asymmetry but, in these cases, the asymmetry is due to bulging on the side of the pathology which can usually be suspected and localized by the neurologic signs. Unilateral coronostenosis, with the exception of eye signs, is not associated with abnormal neurologic findings.

A palpable ridge at the margins of the coronal suture and immobility of the adjacent frontal and parietal bones are additional clinical signs of coronostenosis; mental retardation is commonly found in bilateral coronostenosis but is infrequent in the unilateral form.[3,8] Congenital abnormalities are often found in association with coronostenosis[4,7,12,13,19] and include syndactylism, congenital heart disease, meningocele, cleft palate, micrognathia as the more common varieties. No associated congenital abnormalities were noted in the presented cases or mentioned in those cases of unilateral involvement reviewed in the literature. However, only 2 of the published cases gave summaries of the history and clinical findings.

Facial asymmetry may occur and, when present, varies from a "bashed in" appearance[6] to slight elongation of the face on the involved side. There are other causes of facial asymmetry, including progressive facial hemiatrophy (Romberg's syndrome).[2,10,15] This entity is not associated with skull asymmetry and it occurs later.

Treatment. Wide resection of the pericranium followed by linear craniectomy, producing channels 1.0 to 1.5 cm. wide and parallel to the prematurely fused suture, is the surgery of choice; the bone edges are encased in tantalum foil, acrylic polyethylene tubing, or polyethylene film,[1] the latter being the substance of choice at present. Anderson and Johnson[1] recommend application of Zenker's acetic fixative solution to the exposed dura mater to delay regeneration of bone. When fusion of the bones of the orbit has resulted in exophthalmos, resection of the roof and lateral orbital wall is advised in addition.[12] The squamosal suture should also be visualized and, if fusion has also occurred here, then subtemporal decompression or extension of the craniectomy posteriorly along the squamosal suture has been recommended.

The danger of surgery in proper hands is small relative to the risks of blindness and of psychologic effects secondary to the deformity.[19] A difference of opinion

exists regarding surgical intervention, however, and some[3,8] argue that prophylactic surgery is unnecessary unless progress roentgenograms show extension of the synostosis to involve other sutures or unless cerebral symptoms develop.

Prognosis. In the experience of those with large series of unilateral coronostenosis, this deformity is not accompanied by any brain disturbances.[8] However, not all the sutures undergo closure at the same time in craniostenosis, and subsequent development of premature synostosis in other sutures, particularly in unilateral coronostenosis, is especially likely.[8,19] Patients with unilateral coronostenosis must therefore be closely observed by periodic examinations since brain damage and mental retardation occur frequently when coronal synostosis is bilateral.

Surgical intervention can reduce exophthalmos and strabismus and can prevent blindness. Progressive cosmetic deformity is prevented by the surgery and, as skull growth continues, these deformities tend to correct themselves.

Following craniectomy, there is a tendency for the surgically formed sutures to close within four to eight months, necessitating reoperation in many.[8] Use of the polyethylene film to cover the bone edges has obviated reoperation in most cases but, occasionally, its use is ineffectual.[11] Anderson and Johnson[1] have found that use of polyethylene film delays closure of the surgical sutures for two years or more, and application of Zenker's acetic solution to the dura at the operative site was found to be "probably even more effective."

ROENTGENOLOGIC DISCUSSION

The roentgenologic signs of premature, unilateral coronostenosis include flattening of the frontoparietal and posterior parietal regions; narrowing of the coronal suture, sometimes with sclerosis; elevation of the ipsilateral lesser sphenoid wing and superior margin of the greater sphenoid wing; elliptical orbit enlarged in the coronal plane but diminished in depth on the involved side. The anterior fossa on the ipsilateral side is shallow and shortened and associated with enlargement and deepening of the middle and posterior fossae and depression of the petrous ridge. Displacement of the lambda to the involved side and obliquity of the nasal septum, directed superiorly toward the side of the involved suture, are prominent findings. Although the infant's features may show a marked asymmetry, true asymmetry of the facial bones is either uncommon or difficult to demonstrate roentgenographically. The clinical appearance most likely results from depression of the temporomandibular fossa second to depression of the floor of the middle fossa and consequently of the mandible on the involved side. Thinning of the affected area of the calvarium occurs occasionally. Increased convolutional markings on the same side may be seen in older patients in whom the process is of longer duration, but these are not ordinarily present in infants.

Roentgenograms of the 3 cases presented and of other cases of unilateral coronostenosis reproduced in the literature (Table 1) reveal that certain roentgenographic features (elliptically shaped orbit, elevation of the lesser sphenoid wing, flattening of the parietal region, shortening of the anterior fossa, and enlargement of the middle and posterior fossa) are present in all cases that could be analyzed for these signs. The obliqued nasal septum was found in all but 1 case. Displacement of lambda toward the involved side was a conspicuous finding in 2 cases reported here and in 4 of 6 published cases in which the lambda was demonstrable. The coronal suture was not visualized satisfactorily in 1 of our 3 cases, and the roentgenograms of cases reproduced in the literature usually included only anteroposterior or posteroanterior projections, views in which the coronal suture is not well demonstrated. Of course, when premature narrowing of one of the coronal sutures is shown, this is *prima facie* evidence of premature synostosis, but narrowing of the suture in the absence of marginal sclerosis may be difficult

to recognize or to demonstrate roentgenographically. Nevertheless, even prior to closure of the suture or in the absence of coronal suture visualization, the diagnosis of unilateral coronostenosis may be made with confidence by means of roentgenographic signs which are consistently present.

On being confronted with the skull roentgrams in a case of unilateral coronostenosis for the first time, the possibilities that may come to mind include cerebral hemiatrophy, chronic or recurring subdural hematomas, neurofibromatosis, a slowly growing brain tumor, space-occupying lesions of the orbit, developmental defects of the orbital wall,[14,16] postural asymmetry of the skull, and scoliosis capitis. Analysis of the roentgenograms in these diseases reveals that, although there are several features, including unilateral elevation of the sphenoid wings and asymmetry of the parietal area, which suggest coronostenosis, certain other findings are not consistent with this diagnosis.

On the involved side in chronic or relapsing subdural hematoma, subdural hygroma, and other large, *chronic, space-occupying lesions* of the brain, the skull asymmetry is due to bulging of the ipsilateral temporoparietal region instead of flattening. There may be unilateral bulging of the middle fossa anteriorly, and the sphenoid wings may be pushed up by the same process, but the orbital appearance typical of coronostenosis is absent. The petrous ridge may be elevated in chronic subdural hematoma (when chronic hemiatrophy develops secondarily) and depressed in large, slowly growing brain tumors. Bone destruction in the sella turcica or orbit may be present, especially in neurofibromatosis, and this helps to exclude coronostenosis. Obliquity of the nasal septum is much less marked and much less common in the space-occupying group and, when it occurs, the deviation is more often away from the lesion.

In unilateral coronostenosis as opposed to *cerebral hemiatrophy*, there is absence of asymmetric thickening of the calvarium (and sometimes there is thinning), the petrous ridge is not elevated (and is usually depressed instead), and elevation of the lesser sphenoid wing is much more marked. Shortening of the anterior fossa and diminished orbital depth are not features of cerebral hemiatrophy. Although premature synostosis of the coronal suture probably can occur as a result of hemiatrophy of the brain, it is a secondary process which is rare; the appearance suggesting synostosis in these cases is almost always due to narrowing and overlap of sutures instead.[7] Asymmetry of the facial bones on the same side may occasionally occur with unilateral coronostenosis but has not been described in hemiatrophy. Obliquity of the nasal septum, commonly found in unilateral coronostenosis, makes a considerably less frequent and much less pronounced appearance in cerebral hemiatrophy. These roentgenographic differences also apply to porencephalic cyst except that, in the latter, a local area of thinning or bulging of the calvarium adjacent to the cyst may be present.

Postural asymmetry of the skull and *scoliosis capitis* should present no difficulties in the differential diagnosis, particularly if the clinical history is known.

Congenital absence of the superior orbital wall can give an abnormal appearance to the bony orbit somewhat suggestive of unilateral coronostenosis but has certain features absent in the latter; a portion of the bony orbit is missing, the orbit does not have the typical elliptical appearance, there is always bulging of the temporal fossa, a temporal bone hiatus is present, and abnormality of the sella turcica, which is sometimes deep and sometimes wide and shallow, may be found.[14] Minor unilateral elevation of the lesser sphenoid wing has been noted to occur in *unilateral agenesis* of the eye; this may resemble coronostenosis but the involved orbit is small as opposed to the enlargement usually noted in coronostenosis, and the other signs of unilateral coronostenosis are missing.

Orbital tumors, including neurofibromatosis, and *orbital arteriovenous aneurysms* occasionally cause enlargement of the bony orbit and may cause elevation of the sphenoid wings but, in these, the orbital appearance of coronostenosis is absent, the optic foramen may be enlarged and there may be destruction of the bony orbit.

The typical elliptical shape of the bony orbit in coronostenosis, which Hope[6] likens to the appearance of women's modernistic eyeglasses, is formed by the elevation of the upper, outer corner of the orbit. The superior and lateral orbital margins are straightened and slanted superiorly and laterally; the orbital depth is decreased while the orbit, projected in posteroanterior or anteroposterior views, usually appears enlarged. This appearance was present in all cases of coronostenosis that could be analyzed and is a pathognomonic feature. It is not a finding of any other disease.

In most cases of unilateral coronostenosis, the nasal septum, although ordinarily straight, is seen to lie in oblique position with its superior end deflected towards the synostosis. A similar appearance may occur in any case of craniostenosis with asymmetry of the skull, as in microcephaly and oxycephaly when one coronal suture closes before the other. It is a seldom and minimal finding in cerebral hemiatrophy, porencephaly and the Sturge-Weber disease where the deviation is towards the lesion, and in chronic or relapsing subdural hematoma in which slight deflection of the septum is more often in the opposite direction from the side of the pathology. When marked, as in Case 1, obliquity of the nasal septum is found only in plagiocephaly.

Asymmetry of the face is clinically evident in most cases of unilateral coronostenosis and, although synostosis of the facial bones on the same side occasionally might explain this, depression of the ipsilateral temporomandibular fossa and consequent asymmetric displacement of the mandible is probably the far more frequent reason. That there are some instances of facial asymmetry secondary to true synostosis of the facial bones was suggested by the roentgenograms in Case x, Table 1. In some, the clinical asymmetry may be more apparent than real, an optical illusion due to the unilateral flattening in the frontal region.

Although both are found in unilateral coronostenosis, there is no evidence for any direct cause and effect relationship between septal deviation and facial bony synostosis. This tends to be confirmed by the absence of nasal septum obliquity in progressive facial hemiatrophy as seen in 3 cases reviewed in the literature.[2]

MECHANISM OF THE ANATOMIC CHANGES

The abnormalities of the skull which characterize unilateral coronostensosis may be explained on the basis of knowledge that growth of the bones of the skull occurs at the sutural margins and depends on the expanding effect of the growing brain without which the bone growth will cease.

When premature synostosis occurs at the coronal suture, there is cessation of growth of the parietal and frontal bones at right angles to this suture. Growth continues at uninvolved margins of these bones and other cranial bones apparently with a compensating increase. This results in flattening of the cranium in the frontal and posterior parietal regions of the involved side. The frontal flattening is the more pronounced. While the affected parietal and frontal bones are shortened, their opposite numbers are not, so that lambda and the posterior sagittal suture are displaced to the shorter side of the skull.

The elevation of the lesser sphenoid wing and the superior margin of the greater sphenoid wing and orbital roof is due to the same process. In addition, synostosis may occur at the squamous suture and its extensions, the sphenoparietal and the sphenofrontal sutures, so that the elevation of the lateral margin of the orbit and the sphenoid areas is accentuated.

The nasal septum articulates with the sphenoid bone and any alteration in growth

of the sphenoid must involve this bone. Displacement of the nasal septum towards the side of the synostosis would therefore be expected in conjunction with the deviation of the sphenoid.

The stimulus of the rapidly enlarging brain results in increased bone growth at the uninvolved sutures, and this effect is most pronounced at those sutures near the site of the coronostenosis. It represents a compensatory mechanism which explains the enlarged middle and posterior fossae, depression of the petrous ridge and the absence of significant brain damage.

Modeling due to osteoclastic and osteoblastic activity secondary to pressure exerted by the expanding brain could account for thinning of the affected bone.

Facial asymmetry is apparently the result of enlarging and deepening of the ipsilateral middle fossa which results in displacement inferiorly of the mandibular condyle on the involved side. True unilateral synostosis of the facial bones must be rare.

SUMMARY

CLINICAL SIGNS OF UNILATERAL CORONOSTENOSIS

1. Asymmetric flattening of the posterior parietal and frontoparietal areas.
2. Ridging at margins of involved coronal suture and immobility of adjacent skull bones.
3. Unilateral exophthalmos and divergent strabismus.
4. Asymmetry of the face.
5. Mental retardation in bilateral coronostenosis, but infrequently in unilateral involvement of the coronal suture.
6. Often associated other congenital abnormalities.
7. Appearance of clinical signs in first year of life.

ROENTGENOGRAPHIC SIGNS OF UNILATERAL CORONOSTENOSIS

1. Narrowing of the involved coronal sutures, sometimes with sclerosis.
2. Elliptical, shallow bony orbit, enlarged in the coronal plane, on the same side.
3. Elevation of the ipsilateral lesser sphenoid wing.
4. Flattening of the calvarium, and sometimes thinning, on the side of the synostosis.
5. Shortening of the anterior fossa of skull associated with enlargement of the middle and posterior fossa on the involved side.
6. Depression of petrous ridge on the ipsilateral side.
7. Obliquity of nasal septum directed superiorly toward the side of the involved coronal suture.
8. Deviation of lambda to the same side.
9. Asymmetric position of the mandible and, rarely, asymmetry of the facial bones.

DIFFERENTIAL DIAGNOSIS OF UNILATERAL CORONOSTENOSIS

A. Disease entities most difficult to exclude clinically (these all develop at a later age):

1. Cerebral hemiatrophy and porencephalic cysts.
2. Space-occupying brain lesions including subdural hematomas and brain tumors.
3. Space-occupying lesions of the orbit (tumors and aneurysms).
4. Defective development of the bony orbit.
5. Facial hemiatrophy (Romberg's syndrome).

B. Roentgenographic signs of unilateral coronostenosis as aids in differential diagnosis of these diseases:

1. *Elliptical orbit*, associated with flattening of the frontoparietal or posteroparietal region and elevation of the lesser sphenoid wing on the same side, rules out all other diseases.
2. *Flattening of the parietal bone*, when it occurs with ipsilateral lesser sphenoid wing elevation, excludes all other diseases except possibly cerebral hemiatrophy. In the presence of an elliptical orbit, cerebral hemiatrophy can also be ruled out.
3. *Marked obliquity of a straight nasal septum* is not seen in any other disease, al-

though mild degrees of obliquity sometimes occur in cerebral hemiatrophy and subdural hematoma.

4. *Enlarged middle fossa* permits, in addition to unilateral coronal synostosis, consideration only of space-occupying lesions of the brain (tumors, aneurysms, and chronic and relapsing subdural hematomas).

5. *Depression of the petrous ridge* excludes all other lesions except brain tumors and the rare case of huge aneurysm.

6. *Elevation of the lesser sphenoid wing* may occur in all the diseases listed above under "A" with the exception of progressive facial hemiatrophy.

7. *Facial asymmetry* (but not obliquity of nasal septum or asymmetry of skull) is found in progressive facial hemiatrophy.

8. *Displacement of lambda* in the degree evident in these cases of coronostenosis and in other forms of plagiocephaly is not seen in other conditions.

(Note: unilateral synostosis of the lambdoidal suture can give the same findings except for the elliptical orbit and elevated sphenoid wing.)

CONCLUSIONS

1. Three cases of premature, unilateral synostosis of the coronal suture have been presented along with an analysis of those roentgenograms reproduced in the literature, and this entity is discussed particularly from a standpoint of clinical and roentgenologic diagnosis.

2. When surgical treatment is indicated, early diagnosis is necessary to permit intervention before the complications of craniostenosis are permanently established.

3. Early recognition of this condition is a roentgenologic responsibility.

4. When exophthalmos occurs with frontoparietal and posterior parietal flattening, the clinical evidence is strongly suggestive of unilateral coronostenosis. Nevertheless, cerebral hemiatrophy can be differentiated with certainty only by roentgenographic examination.

5. Narrowing of the coronal suture, when present, is *prima facie* evidence of coronostenosis, but this may be absent early in the disease or difficult to demonstrate. However, an enlarged, elliptical orbit (as seen on posteroanterior and anteroposterior roentgenograms) is always present and is a pathognomonic sign. The correctness of the diagnosis may be confirmed by the other roentgenographic signs on the ipsilateral side, especially the following: (a) elevated sphenoid wing and (b) flattened parietal bone.

6. Obliquity of the nasal septum is a helpful adjunctive sign which, when of marked degree, has been found only in plagiocephaly. Out of 10 cases of coronostenosis that could be analyzed for this sign, it was present in 9.

7. Definite displacement of lambda to the involved side has not been noted in conditions other than plagiocephaly.

M. H. Nathan, M.D.
Jefferson Davis Hospital
Houston 3, Texas

The authors wish to express their gratitude to Dr. Ezra Neidich and to Dr. Edward Singleton for permitting us the use of Case II and Case III respectively. The kindness of Dr. Frederic Silverman and Dr. Harris Jackson in permitting one of the authors (M.H.N.) the use of reprints from their personal reprint files is greatly appreciated as was certain information offered by Dr. John W. Hope.

REFERENCES

1. ANDERSON, F. M., and JOHNSON, F. L. Craniosynostosis; modification in surgical treatment. *Surgery*, 1956, *40*, 961–970.
2. ARCHAMBAULT, L., and FROMM, N. K. Progressive facial hemiatrophy; report of 3 cases. *Arch. Neurol. & Psychiat.*, 1932, *27*, 529–584.
3. BERTELSTEN, T. I. Premature synostosis of cranial sutures. *Acta ophth.*, 1958, Suppl. 51.
4. CAFFEY, J. Pediatric X-Ray Diagnosis. Third edition. Year Book Publishers, Chicago, 1956, pp. 36–44.
5. HAAS, L. L. Roentgenological skull measurements and their diagnostic applications. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1952, *67*, 197–209.
6. HOPE, J. W. Personal communication.
7. HOPE, J. W., SPITZ, E. B., and SLADE, H. W. Early recognition of premature cranial synostosis. *Radiology*, 1955, *65*, 183–193.

8. INGRAHAM, F. D., ALEXANDER, E., JR., and MATSON, D. D. Clinical studies in craniostenosis; analysis of 50 cases and method of surgical treatment. *Surgery*, 1948, *24*, 518–541.
9. JACKSON, H. Asymmetry and growth of skull. *Brit. J. Radiol.*, 1956, *29*, 521–535.
10. JACOBS, E. M. Progressive facial hemiatrophy. *Neurology*, 1955, *5*, 444–446.
11. LAITINEN, L., MIETTINEN, P., and SULAMAA, M. Ophthalmological observations in craniosynostosis. *Acta ophth.*, 1956, *34*, 121–132.
12. MCLAURIN, R. L., and MATSON, D. D. Importance of early surgical treatment of craniosynostosis; review of 36 cases treated during first six months of life. *Pediatrics*, 1952, *10*, 637–652.
13. PENDERGRASS, E. P., SCHAEFFER, J. P., and HODES, P. J. The Head and Neck in Roentgen Diagnosis. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1956, pp. 121–131.
14. ROBERTSON, E. G. Pulsating exophthalmos due to defective development of sphenoid bone. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *62*, 44–51.
15. RUSHTON, M. A. Early case of facial hemiatrophy. *Oral Surg.*, 1951, *4*, 1457–1460.
16. SCHWARTZ, C. W., and COLLINS, L. C. The Skull and Brain Roentgenologically Considered. Charles C Thomas, Publisher, Springfield, Ill., 1951, pp. 75–76.
17. SEAMAN, W. B., and FURLOW, L. T. Anomalies of bony orbit. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1954, *71*, 51–59.
18. SEAR, H. R. Some notes on craniostenosis. *Brit. J. Radiol.*, 1932, *10*, 445–487.
19. SILVERMAN, F. N. Premature synostosis of cranial sutures; review. *Ohio M. J.*, 1954, *50*, 131–137.

VOL. 86, No. 3

SKULL CHANGES IN IRON DEFICIENCY ANEMIA SIMULATING CONGENITAL HEMOLYTIC ANEMIA*

By HENRY BURKO, M.D.,† HARRY Z. MELLINS, M.D.,† *and* JANET WATSON, M.D.‡
BROOKLYN, NEW YORK

WIDENING of the diploic space and thinning of the outer table of the skull, originally described in children with chronic hemolytic anemia, were reported for the first time in a child without hemolytic anemia or red blood cell sickling by Feingold and Case[7] in 1933. A museum study of the skulls of ancient Peruvians, by these authors, revealed that the incidence of similar skull changes was far greater than the anticipated incidence of hemolytic disease. The relationship between these skull changes and iron deficiency anemia was first indicated by Eng[5] in 1958. He reported cortical thinning and diploic widening in the frontal and parietal bones of a twelve year old girl, and published the results of an extensive examination of her blood. Hemoglobin analysis revealed a normal amount of alkali-resistant hemoglobin and no abnormal hemoglobin. There was, however, severe hypochromic microcytic anemia and erythroid hyperplasia of the sternal marrow, with iron depletion. It was presumed that similar marrow hyperplasia occurring in the skull produced the changes seen roentgenographically. The patient felt well after treatment with iron preparations and the hematologic indices returned to normal. In 1960, Shahidi and Diamond[11] reported 3 cases of iron deficiency anemia with skull changes simulating the hemolytic anemias, and, later the same year, Britton, Canby, and Kohler[2] added 5 cases to the literature. This report will describe the skull changes in 7 patients with iron deficiency anemia recently seen at the Kings County Hospital Center.

REPORT OF CASES

CASE I (Fig. 1, *A* and *B*). A Negro girl, nineteen months old, was admitted to the hospital because of coryza, cough, and fever of three days' duration. She had been born at term weighing six pounds seven ounces and was one of twins. At the time of admission, she was pale and malnourished, her face was puffy and there were prominent bulges over the frontal and parietal bones. The liver and spleen were enlarged.

Examination of the blood revealed a microcytic hypochromic anemia with a hemoglobin concentration of 3.5 gm./100 ml. Skull roentgenograms showed widening of the diploic space in the frontal and parietal bones, trabecular atrophy and thinning of the tables. She received transfusions of packed red blood cells and was sent home.

CASE II (Fig. 2). A Negro girl, thirteen months old, was admitted in acute respiratory distress, with a cough and fever. She had been born with a twin at term weighing four pounds fifteen ounces. Gestation had been complicated by frequent maternal episodes of vaginal bleeding resulting in significant maternal anemia. The child's diet had contained milk and solids but was devoid of meats and nutritional supplements.

On admission, the child was pale and had an enlarged liver. Hemoglobin concentration was 4.0 gm./100 ml. Skull roentgenograms revealed widening of the diploic space in the frontal bone, with trabecular atrophy.

CASE III (Fig. 3, *A* and *B*). A Negro boy, who had been born at seven months' gestation weighing three pounds six ounces, had been fed exclusively with evaporated milk formula. At twelve months of age, the child was taken to a

* From the Departments of Radiology and Pediatrics of the State University of New York Downstate Medical Center and the Kings County Hospital Center, Brooklyn, New York.
† Department of Radiology.
‡ Department of Pediatrics.

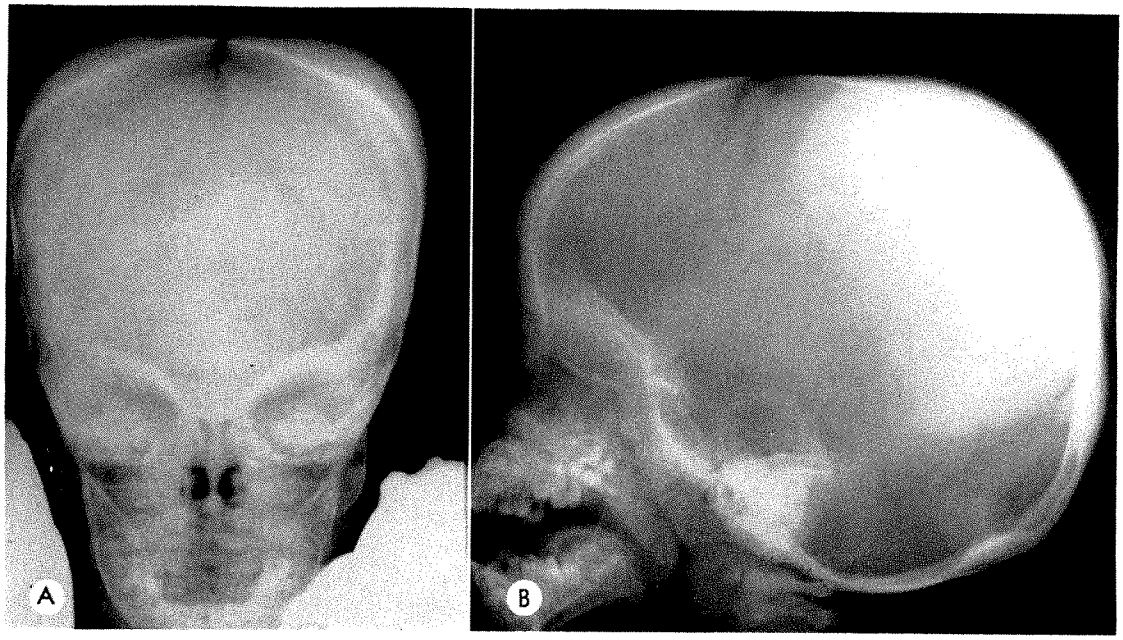


FIG. 1. Case I. (*A* and *B*) Skull roentgenograms reveal marked trabecular atrophy with thinning of the tables and widening of the diploic space. Changes are most marked in the parietal bones, in which vertical striations are noted.

health station for a periodic examination. Following the advice of the examiner, the child was brought to the hospital.

At the time of admission, the child was pale and underdeveloped. His eyelids and legs were puffy. His head was distorted by prominences in the parietal regions.

Hemoglobin concentration on admission was 3 gm./100 ml. Roentgenograms of the skull demonstrated a widened diploic space in the frontal and parietal bones, with atrophy of the outer table in the medial part of the midparietal bones.

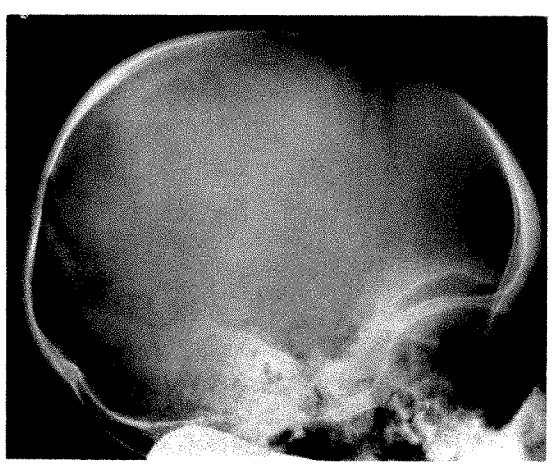


FIG. 2. Case II. Lateral view of the skull reveals trabecular atrophy in the frontal bone, with widening of the diploic space.

CASE IV. A Negro boy, aged fourteen months, was admitted to the hospital because of respiratory distress and coryza of two days' duration. He had been born at term weighing four pounds four ounces. Evaporated milk formula comprised his diet almost exclusively.

On admission the child was pale and underweight and had an enlarged liver. Hemoglobin concentration was 1.4 gm./100 ml. Skull roentgenograms revealed widening of the diploic space in the frontal and parietal bones, with mild trabecular atrophy and thinning of the outer tables.

CASE V. A twenty-four month old Negro boy was brought to the emergency room for treatment of an insect bite and was admitted to the hospital because of pallor. The child had been born at term weighing six pounds. He had been fed milk exclusively since birth. Hemoglobin concentration was 3.3 gm./100 ml. Skull roentgenograms revealed widening of the diploic space and thinning of the outer table of the parietal bones.

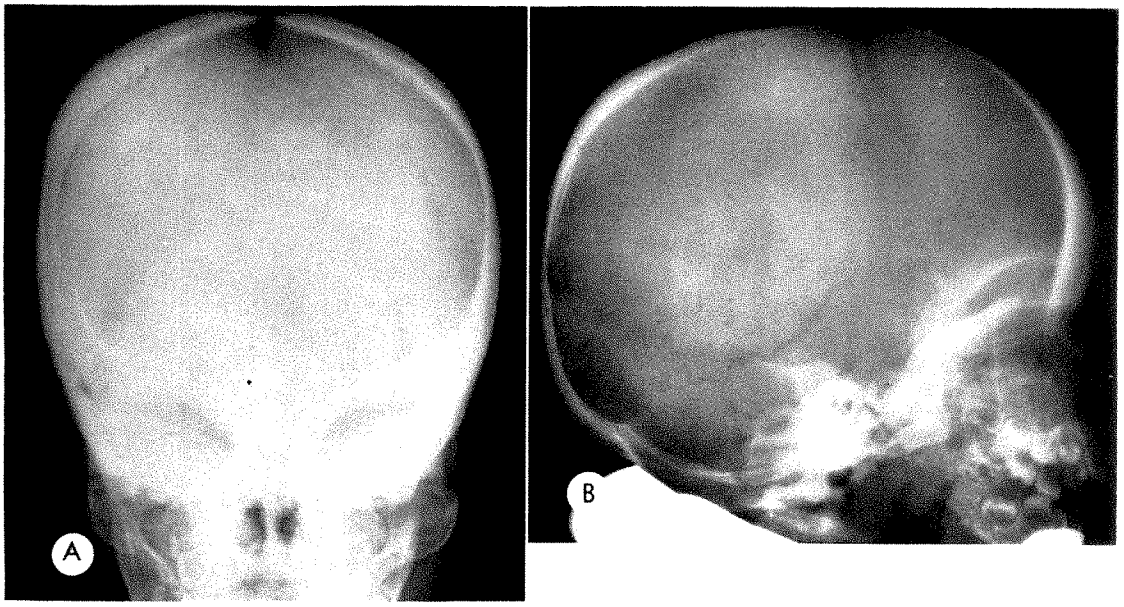


FIG. 3. Case III. (*A* and *B*) Skull roentgenograms reveal the widened diploic space in the frontal and parietal bones with considerable thinning of the outer table in the medial part of the parietal bones.

CASE VI. A Negro girl, ten months old, was admitted because of fever and cough. The child had been born at term weighing six pounds and three ounces. Her diet consisted of evaporated milk formula.

On admission, the child was underweight and underdeveloped. She was unable to sit without support.

Hemoglobin concentration was 7.8 gm./100 ml. Skull roentgenograms revealed a widened diploic space, marked thinning of the outer table, and trabecular atrophy involving the parietal bones to a greater extent than the frontal bones.

CASE VII. A Negro boy, aged twenty-four months, was admitted because of a scalp and ear infection. A birth and dietary history could not be obtained. The child had been abandoned by his parents and was being cared for by a neighbor.

Hemoglobin concentration on admission was 6.5 gm./100 ml. Skull roentgenograms revealed a widened diploic space in the frontal and parietal bones.

The diagnosis of chronic iron deficiency anemia in these 7 patients was established by the demonstration of hypochromic microcytic red blood cells and by the subsequent rise in hemoglobin concentration following iron therapy.

REVIEW OF ALL REPORTED CASES OF IRON DEFICIENCY ANEMIA WITH SKULL CHANGES

Comparisons among the 16 reported patients with iron deficiency anemia and skull changes may be summarized as follows:

Birth History. Half of the children were born prematurely; half were born with a twin.

Diet. A majority of the children had a dietary intake restricted to milk.

Physical Findings. Almost all of the children were poorly developed and poorly nourished. Many had palpable cranial bossing. Half of the children had moderate enlargement of the liver and spleen. Two children had edema of the face and eyelids.

Hematologic Considerations. The hemoglobin concentration, before therapy, ranged from 1.4 to 7.8 gm./100 ml. Serum iron determinations (not performed in patients reported in this paper) were below 50 μgm./100 ml. No evidence of increased erythrocyte destruction, abnormal hemoglobins, or increase in alkali-resistant hemo-

globin was found. Bone marrow studies, when performed, revealed marked erythroid hyperplasia and increased iron binding capacity.

Roentgen Findings. The long bones examined roentgenographically were found to be normal.

DISCUSSION

Marrow hyperplasia is a striking manifestation of the chronic hemolytic anemias. The anatomic structures responsible for the roentgen appearance of marrow hyperplasia have been carefully studied. Cooley, in 1927,[10] and, subsequently, Caffey[3] described necropsy findings in thalassemia major. The diploic spaces of the calvarium are widened and filled with "sponge-like marrow traversed by bony trabeculae running at right angles to the tables." Both tables are atrophic, particularly the outer table. The calvarium, the mandible, and the facial bones, as well as the axial skeleton and appendages, are similarly involved. Cooley's explanation of the bone changes is that they are "the result of the reaction of marrow to prolonged overstimulation as a consequence of chronic hemolysis, beginning before the cortex is too firm to permit overgrowth of marrow."[3]

The roentgen appearance of marrow hyperplasia results from the expansion of young bone by an overstimulated marrow. In the first few weeks of normal life, erythropoiesis is relatively inactive.[6] As a consequence, the hemoglobin level in a normal newborn begins to fall soon after birth and may reach a minimum level in one to two months.[10] The relative hypoxia, and other stimuli, initiate active erythropoiesis. In the presence of anemia, the erythropoietic response is exaggerated and the degree of response is, in general, related to the severity of the anemia. The overactive erythropoiesis is manifested largely as marrow hyperplasia. A latent period of several months occurs between the clinical onset of anemia and the roentgen evidence of marrow hyperplasia. Caffey[3] has described the roentgen appearance of marrow hyperplasia as early as three months after the onset of anemia.

Roentgen evidence of marrow hyperplasia occurs when severe anemia has an early onset. The age of patients at the time of the initial appearance of congenital hemolytic anemias varies somewhat. Several congenital hemolytic syndromes, such as thalassemia major and congenital spherocytosis, may be present at, or shortly after, birth.[6] On the other hand, hemolytic anemias characterized by the production of an abnormal type of hemoglobin are delayed in onset by the presence of large amounts of fetal hemoglobin. Sickle cell anemia is a prime example of this latter type of hemolytic anemia, with the onset of anemia usually occurring after six months of age.

Iron deficiency anemia may occur early in life and, in many instances, is severe. It is defined as a transition from a normocytic, normochromic "blood picture" to one that is microcytic, hypochromic, and it responds to treatment with iron.[13] Infants and growing children require iron for increase in blood volume and myoglobin volume, and for respiratory enzymes which are required by all cells. It is felt that normal growing children maintain only a slightly positive iron balance. Inadequate iron in the diet or inadequate absorption of iron can readily result in iron deficiency.[8] In the absence of adequate exogenous iron sources or adequate iron absorption, the exhaustion of endogenous iron reserve can occur two to three months after birth,[10] resulting in the onset of iron deficiency anemia.

Several clinical investigations have emphasized the predisposing factors in iron deficiency anemia. In a study of 272 cases of iron deficiency anemia, Woodruff[12] found a birth weight distribution which was considerably less than average, with a high incidence of twins and premature births. Dawson and Desforges[4] also found a predominance of premature infants and twins. In addition, maternal iron depletion and inadequate diet appeared to play major

roles in the production of iron deficiency anemia. In over half the cases reviewed, two or more of the above factors contributed to anemia. In instances where one factor was important, this was most frequently a deficient diet. In a review of antenatal factors contributing to infant blood disorders, Potter[9] cites anemia from blood loss *in utero*, and anemia from blood destruction by infections and antibodies. These blood losses may increase the severity of subsequent iron deficiency anemia. The above studies indicate that low birth weight, most profound in the presence of twinning and prematurity, results in a relatively greater growth rate and nutritional requirement. Antenatal loss of iron, either by maternal or fetal iron depletion, reduces endogenous iron stores. In the absence of these predisposing factors, a diet deficient in iron appears to be the most important cause of iron deficiency anemia.

Caffey[3] has described the skeletal changes in thalassemia major in comparison with those in other chronic hemolytic anemias. The earliest change in the skull is described as widening of the diploic space—initially, in the inferior squamosal portion and horizontal plate of the frontal bone and, subsequently, in the parietal bone. Diffuse osteoporosis and trabecular atrophy occur, with thinning of the tables, more marked in the outer table. Later, coarse trabeculae, oriented as vertical striations, appear in the posterior frontal and anterior parietal bones.

As a rule, the most severe clinical cases develop the earliest and most marked bone lesions. However, in patients of the same age, with similar clinical and hematologic findings, surprising differences in the roentgen findings in the skull are encountered. Caffey[3] concluded that in thalassemia major "the skull alone is not necessarily a reliable criterion of the presence of (hemolytic) disease, nor is it an index of the degree of involvement of the remainder of the skeleton." In other chronic hemolytic anemias the changes in the skull vary even more widely. In many instances, the roentgen findings are identical with the early changes described in thalassemia major—widening of the diploic space in the frontal bone with diffuse osteoporosis and trabecular atrophy; occasionally, however, the parietal bones are involved to a greater extent than the frontal bones.

The reported patients with skull changes in chronic iron deficiency anemia have demonstrated the same variability in appearance as in the chronic hemolytic anemias. A comparison of the skull roentgenograms reproduced in this paper reveals considerable variation in the severity of bony changes as well as differences in the sites of most marked involvement. Siblings of the children, many of whom had equally severe iron deficiency anemia, had no skull changes. Many children admitted to the Kings County Hospital Center with profound iron deficiency anemia had no discernible skeletal abnormalities. It is evident that in both chronic hemolytic anemia and in chronic iron deficiency anemia the skull is an unpredictable and unreliable index of the presence or severity of anemia.

Aside from marrow hyperplasia in the chronic hemolytic anemias and chronic iron deficiency anemia, skull changes have been reported in 2 patients with congenital cardiac anomalies associated with cyanosis and polycythemia.[1] Simulators of marrow hyperplasia in the skull[2] are rickets, pseudohyperparathyroidism, infantile cortical hyperostosis, and idiopathic hypercalcemia. No problem should be encountered in clinical differentiation.

SUMMARY

Marrow hyperplasia in young, yielding skull bones causes widening of the diploic spaces and thinning of the outer tables. These roentgen findings were originally attributed to the marrow hyperplasia of chronic hemolytic anemias but they recently have been reported in 9 children with chronic iron deficiency anemia. This report adds 7 children to those reported with skull changes in iron deficiency ane-

mia. Like chronic hemolytic anemias, iron deficiency anemia may occur early in childhood in a severe form. Anemia invokes an exaggerated erythroid response, manifested largely as marrow hyperplasia. The skull changes of marrow hyperplasia vary considerably in different children and do not reflect the severity of anemia.

Henry Burko, M.D.
Department of Radiology
Kings County Hospital Center
451 Clarkson Avenue
Brooklyn 3, New York

REFERENCES

1. ASCENZI, A., and MARINOZZI, V. Sur le crâne en bosse au cours des polyglobulies secondaires à l'hypoxémie chronique. *Acta haemat.*, 1958, *19*, 253–262.
2. BRITTON, H. A., CANBY, J. P., and KOHLER, C. M. Iron deficiency anemia producing evidence of marrow hyperplasia in calvarium. *Pediatrics*, 1960, *25*, 621–628.
3. CAFFEY, J. Skeletal changes in chronic hemolytic anemias (erythroblastic anemia, sickle cell anemia and chronic hemolytic icterus). AM. J. ROENTGENOL. & RAD. THERAPY, 1937, *37*, 293–324.
4. DAWSON, J. P., and DESFORGES, J. F. Dietary and storage factors in iron-deficiency anemia of infancy. *Am. J. Dis. Child.*, 1958, *96*, 169–179.
5. ENG, L. I. L. Chronic iron deficiency anemia with bone changes resembling Cooley's anemia. *Acta haemat.*, 1958, *19*, 263–268.
6. ERLANDSON, M. E., and HILGARTNER, M. Hemolytic disease in neonatal period and early infancy. *J. Pediat.*, 1959, *54*, 566–585.
7. FEINGOLD, B. F., and CASE, J. T. Roentgenologic skull changes in anemias of childhood. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *29*, 194–202.
8. MOORE, C. V. Importance of nutritional factors in pathogenesis of iron deficiency anemia. *Scandinav. J. Clin. & Lab. Invest.*, 1957, *9*, 292–304.
9. POTTER, E. L. The antenatal background of blood diseases of infancy. *J. Pediat.*, 1959, *54*, 552–565.
10. SCHULMAN, I. Anemia of prematurity. *J. Pediat.*, 1959, *54*, 663–672.
11. SHAHIDI, N. T., and DIAMOND, L. K. Skull changes in infants with chronic iron-deficiency anemia. *New England J. Med.*, 1960, *262*, 137–139.
12. WOODRUFF, C. W. Multiple causes of iron deficiency anemia in infants. *J.A.M.A.*, 1958, *167*, 715–720.
13. Iron deficiency anemia; report of study group. *WHO Technical Report Series*, 1959, *182*, 1–15.

HYDRANENCEPHALY: PLAIN FILM FINDINGS*

By LEONID CALENOFF, M.D.†
CHICAGO, ILLINOIS

HYDRANENCEPHALY is an anomaly consisting of complete or nearly complete absence of the cerebral hemispheres with intact meninges and skull.[8] The resulting cavity is filled with cerebrospinal fluid. This condition, an autopsy curiosity a few years ago, is being diagnosed now during life by means of transillumination of the skull[7] and special roentgenologic procedures such as ventriculography and carotid angiography.

A recent case of hydranencephaly, for which we had only plain skull roentgenograms (Fig. 1 and 2), caused us to review the literature. We could not find any illustrations of plain skull roentgenograms for cases of hydranencephaly, despite the fact that they are diagnostic and that it would be helpful to know the specific findings.

ILLUSTRATIVE CASE

Our case was an infant, delivered with a breech presentation, who cried after birth and who presented a Bell's palsy, because of which skull roentgenograms were obtained two hours after birth. The anteroposterior and lateral views showed normal measurements (head circumference, 35.5 cm.; chest circumference, 37 cm.), thinning of the inner table, undifferentiated external table at the frontoparietal areas, and prominent suture and fontanelle width.

The infant died twenty-four hours later and at autopsy the cerebral hemispheres were found to be missing; they were represented by two small buds of brain tissue lying over the intact pons and cerebellum. The meninges were intact and filled with fluid which was not under pressure.

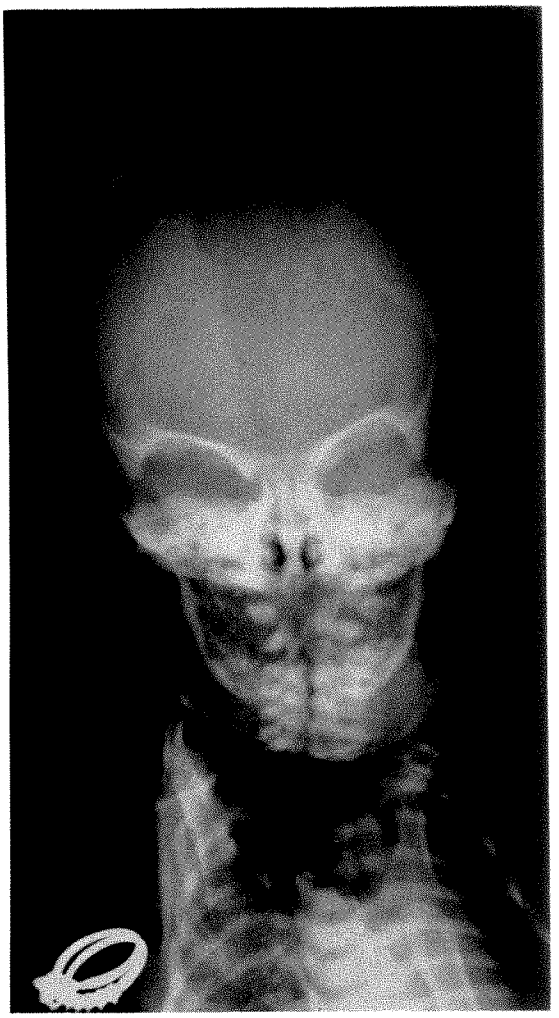


FIG. 1. Anteroposterior roentgenogram of the skull of a newborn infant with proved hydranencephaly. The apparent widening of the suture in the normal-sized skull is due to incomplete ossification of the calvarial bones.

DISCUSSION

In addition to transillumination[3] and flat line electroencephalography,[1] plain skull roentgenography, ventriculography and carotid angiography are the roentgenologic procedures of choice, depending on the degree of underdevelopment of the hemispheres.

In ventriculography,[2] 300 cc. of cerebrospinal fluid is replaced by oxygen and usu-

* From the Department of Radiology, College of Medicine, University of Illinois, Chicago, Illinois.
† Instructor in Radiology.

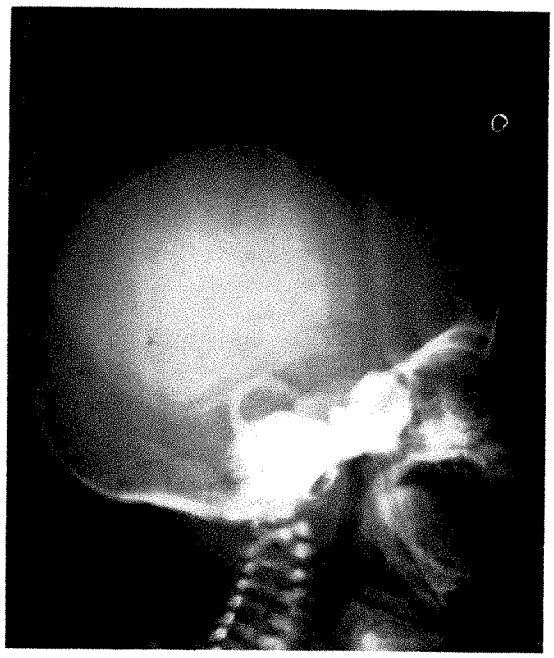


FIG. 2. Lateral roentgenogram of the skull in the same case as in Figure 1. The skull measurements are within normal limits. There are no signs of increased intracranial pressure.

ally an enormous cavity with a fluid level is demonstrated. There is ordinarily no evidence of ventricular pattern or cerebral cortex. Inverted, vertical and lateral views sometimes show residual, nondeveloped cerebral tissue in the posterior fossa or frontal region.

The carotid angiogram[4] usually demonstrates an anterior cerebral artery, essentially normal in position but with slight straightening of the ascending segment. The medial cerebral artery lies closer to the base of the skull and, on the anteroposterior roentgenogram, shows a curvature of the proximal segment with downward convexity.

The findings on the plain skull roentgenogram,[7] the ones we like to stress, are: a normal-sized skull for the infant's age, although sometimes slightly larger[9] or slightly smaller[6] than normal; and incomplete ossification of the calvarial bones suggesting separation of the sutures and widening of the fontanelle—features which, however, are only apparent.

This particular aspect of the skull in hydranencephaly demonstrates a developmental anomaly caused by the lower pressure exerted by the fluid (and meninges) on the skull than the brain would exercise. For the same reason, an anencephalic would not develop a calvarium, and a hydrocephalic would develop thin skull bones, with separated sutures and a marked increase in skull size.

SUMMARY

The roentgen diagnosis of hydranencephaly is discussed briefly in connection with the plain skull roentgenograms of a proved case. No similar roentgen studies are known to have been published. The plain roentgenography skull findings in hydranencephaly are summarized as follows: skull size within normal limits for the infant's age; marked thinning of the inner table, without osteolysis; undifferentiated external table at the frontoparietal areas; apparent widening of sutures and fontanelle and, sometimes, the presence of cystic shadows projecting over the frontoparietal areas and corresponding to the cerebral ventricles.

Department of Radiology
University of Illinois
College of Medicine
840 South Wood Street
Chicago 12, Illinois

Acknowledgment is hereby made for the use of roentgenograms from the X-Ray Department, MacNeal Memorial Hospital, Berwyn, Illinois.

REFERENCES

1. CAFFEY, J. Pediatric X-Ray Diagnosis; A Textbook for Students and Practitioners of Pediatrics, Surgery and Radiology. Fourth edition. Year Book Publishers, Inc., Chicago, 1961.
2. EDGE, W. E. B. Hydranencephaly. *South African M. J.*, 1955, *29*, 470–472.
3. HAMBY, W. B., KRAUSS, R. F., and BESWICK, W. F. Hydranencephaly: clinical diagnosis; presentation of seven cases. *Pediatrics*, 1950, *6*, 371–383.
4. LEFEVRE, A. B., ZACLIS, J., and VALENTE, M. I. Hidrocefalia ou hidranencefalia. Valor da transiluminação do crânio no diagnóstico diferencial. Estudo anátomo-clínico de dois casos.

Arq. neuro-psiquiat., 1955, *13*, 325–337.
5. Muir, C. S. Hydranencephaly and allied disorders; study of cerebral defect in Chinese children. *Arch. Dis. Childhood*, 1959, *34*, 231–246.
6. Najman, E. Case of hydranencephaly diagnosed clinically. *Ann. paediat.*, 1953, *181*, 249–255.
7. Olive, J. T., and DuShane, J. W. Hydranencephaly. *Am. J. Dis. Child.*, 1953, *85*, 43–46.
8. Pendergrass, E. P., Schaeffer, J. P., and Hodes, P. J. The Head and Neck in Roentgen Diagnosis. Vol. II. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1956, p. 1159.
9. Poser, C. M., Walsh, F. C., and Scheinberg, L. C. Hydranencephaly. *Neurology*, 1955, 5, 284–289.

MENINGIOMA OF THE POSTERIOR FOSSA IN A CHILD*

By WILLIAM E. GANNON, M.D.
BROOKLYN, NEW YORK

MENINGIOMAS are tumors rarely seen before adolescence. Bailey *et al.*[1] reported 2 meningiomas in a series of 100 tumors of childhood. Globus *et al.*[6] found 2 meningiomas in a series of 92 tumors of childhood and adolescence, and Cuneo and Rand[3] encountered 2 in a series of 83 intracranial tumors in childhood. Bergstrand *et al.*[2] reported 2 meningiomas among 293 brain tumors in children, Ingraham and Matson[8] found 1 in 313 intracranial tumors in children, and Paillas *et al.*[10] reported 2 among 293 brain tumors in children.

A case of meningioma of the posterior fossa in a child with calcification as a chief finding is being reported.

REPORT OF A CASE

This four year old Puerto Rican female was transferred from another hospital with a history of generalized convulsive seizures of one month's duration. While in the other hospital, she had also developed ataxia. Physical examination upon admission revealed nystagmus on left lateral gaze, left peripheral facial paresis, left facial hypalgesia, and a diminished left corneal reflex. The deep tendon reflexes were hyperactive bilaterally. The finger to nose test was impaired bilaterally but more so on the left.

Roentgenograms of the skull revealed a large area of fine calcification, posterior and inferior to the left petrous bone, best seen on the sagittal roentgenograms (Fig. 1 and 2), which appeared to be made up of radiating strands.

A suboccipital craniotomy disclosed a finely granular, moderately vascular tumor arising from the posterior surface of the left petrous bone, extending almost to the midline and displacing the pons and brainstem to the right. An incomplete removal was performed. The pathologic diagnosis was fibrocytic meningioma.

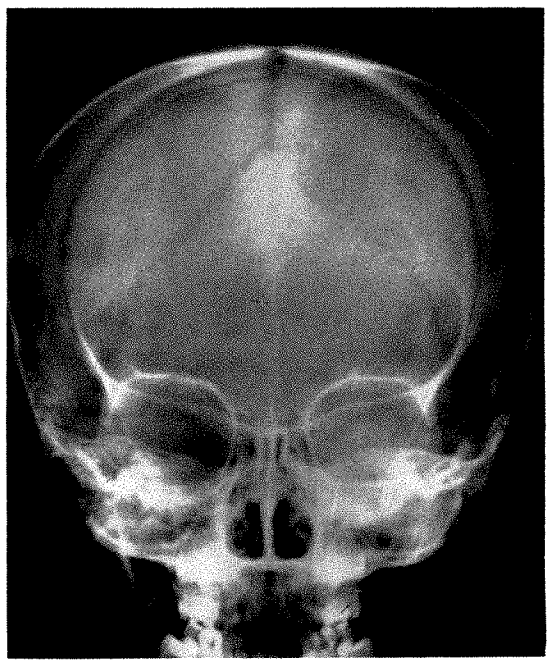


FIG. 1. Posteroanterior roentgenogram showing the area of calcification through the left bony orbit.

DISCUSSION

Meningiomas arising in the posterior fossa are infrequent at any age. Grant[7] found only 3 meningiomas of the posterior fossa in a series of 149 intracranial meningiomas. In Olivecrona's[9] series of 4,185 cases of verified brain tumors, there were 68 cases of meningioma of the posterior fossa, representing 1.7 per cent of all cases. The ratio of posterior fossa meningiomas to all intracranial meningiomas was 1 to 12. Cushing and Eisenhardt[4] found a ratio of 1 to 13 in their series of 295 intracranial meningiomas. They called attention to the difference in area between the subtentorial and supratentorial dura, stating that the

* From the Departments of Radiology of the State University of New York, Downstate Medical Center, Brooklyn, New York, and the Kings County Hospital Center, Brooklyn, New York.

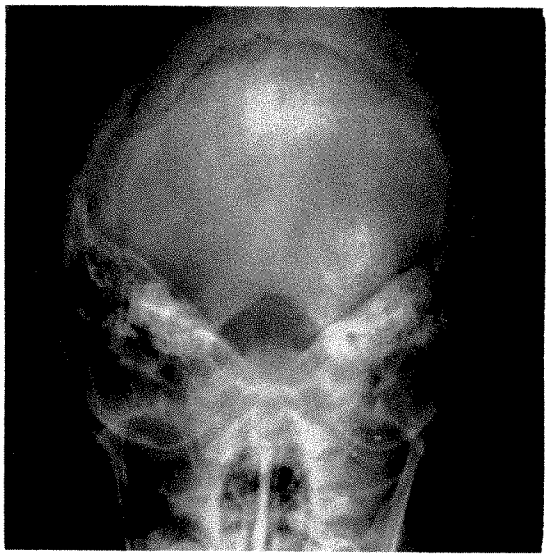


Fig. 2. Occipital roentgenogram showing the calcification arising from the medial two-thirds of the left petrous bone.

subtentorial dura measures 166 sq. cm. while the supratentorial dura measures 754 sq. cm.

In a series of 273 tumors in children French[5] found 7 meningiomas, 2 of them subtentorial. Tristan and Hodes[11] had only 1 patient under ten years of age in 59 cases of posterior fossa meningioma.

Calcification has been frequently described in supratentorial meningiomas in children and in meningiomas of the posterior fossa in adults; however, in a review of several large series of infratentorial meningiomas in children, calcification has not been mentioned as a finding.

SUMMARY

A case of meningioma of the posterior fossa in a child is reported. In this patient marked calcification was noted in the tumor. Calcification in such meningiomas, while fairly common in adults, appears to be an unusual finding in children.

Department of Radiology
State University of New York
Downstate Medical Center
450 Clarkson Avenue
Brooklyn 3, New York

REFERENCES

1. Bailey, P., Buchanan, D. N., and Bucy, P. C. Intracranial Tumors of Infancy and Childhood. University of Chicago Press, Chicago, 1939.
2. Bergstrand, C. G., Bergstedt, J., and Herrlin, K. M. Paediatric aspects of brain tumors in infancy and childhood. *Acta paediat.*, 1958, *47*, 688–705.
3. Cuneo, H. M., and Rand, C. W. Brain Tumors of Childhood. Charles C Thomas, Publisher, Springfield, Ill., 1952.
4. Cushing, H., and Eisenhardt, L. Meningiomas: Their Classification, Regional Behavior, Life History, and Surgical End Results. Charles C Thomas, Publisher, Springfield, Ill., 1938.
5. French, L. A. Tumors—intracranial and cranial. In: Pediatric Neurosurgery. Edited by Jackson, I. J., and Thompson, R. K. Charles C Thomas, Publisher, Springfield, Ill., 1959.
6. Globus, J. H., Zucker, J. M., and Rubenstein, J. M. Tumors of brain in children and in adolescents; clinical and anatomic survey of ninety-two verified cases. *Am. J. Dis. Child.*, 1943, *65*, 604–663.
7. Grant, F. C. Intracranial meningiomas, surgical results. *Surg., Gynec. & Obst.*, 1947, *85*, 419–431.
8. Ingraham, F. D., and Matson, D. D. Neurosurgery of Infancy and Childhood. Charles C Thomas, Publisher, Springfield, Ill., 1954.
9. Olivecrona, H. Cited by Castellano, F., and Ruggiero, G. Meningiomas of posterior fossa. *Acta radiol.*, 1953, Suppl. 104.
10. Paillas, J. E., Vigouroux, R., Piganiol, G., and Sedan, R. Les tumeurs supratentorielles de l'enfant. *Neuro-chirurgie*, 1957, *3*, 165–179.
11. Tristan, T. A., and Hodes, P. J. Meningiomas of posterior cranial fossa. *Radiology*, 1958, *70*, 1–14.

INTRACRANIAL CALCIFICATION IN INFANTS*

IS IT POSSIBLE ROENTGENOGRAPHICALLY TO DISTINGUISH BETWEEN TOXOPLASMOSIS AND CYTOMEGALIC INCLUSION DISEASE?

By ARTHUR S. TUCKER, M.D.
CLEVELAND, OHIO

CALCIFICATIONS of the brain in infants can almost always be considered to be of inflammatory origin. Tumor and physiologic origins of calcified brain tissue are almost unheard of in the first few years of life. Of the inflammatory processes causing intracranial calcification in infants, toxoplasmosis is easily the most common. Hundreds of cases have been reported since the identification of *Toxoplasma* as a human pathogen by Wolf, Cowen, and Paige in 1939.[7]

The most helpful single finding in diagnosing toxoplasmic infection is the presence of chorioretinopathy. Other conditions encountered frequently enough to form with chorioretinopathy a tetrad of diagnostic features are cerebral calcification, hydro- or microcephaly, and psychomotor disturbances. The diagnosis is best substantiated by serologic studies. Of these, the most practicable in most instances is the dye test for antibodies. In some cases, complement fixation is a useful adjunct, although this antibody develops more slowly and disappears more rapidly than the dye test antibody.

The calcifications in toxoplasmosis commonly are in foci scattered through gray and white matter. Apart from a tendency to be more numerous in the posterior than in the anterior part of the brain (Fig. 1 and 2), the distribution of the calcifications covers a wide area. Dyke *et al.*[3] in 1942 pointed out that a most characteristic but, unfortunately, not most common distribution was in curved streaks in the basal ganglia and thalami (Fig. 3).

In recent years, the reports on toxoplasmosis as a cause of intracranial calcification have been outnumbered by those describing a new disease of virus etiology commonly called cytomegalic inclusion disease. In this infection of the newborn child, there always is jaundice, and the clinical manifestations, particularly as related to the eyes and central nervous system, may be indistinguishable from those described in toxoplasmosis. Intracranial calcifications may be present and, as in toxoplasmosis, the lesions are apt to be multiple scattered foci. Other organ systems of the body are commonly involved, and the liver and spleen invariably show severe effects of the infection. The diagnosis is frequently made by finding inclusion cells in the urine, gastric washings, or other secretions.

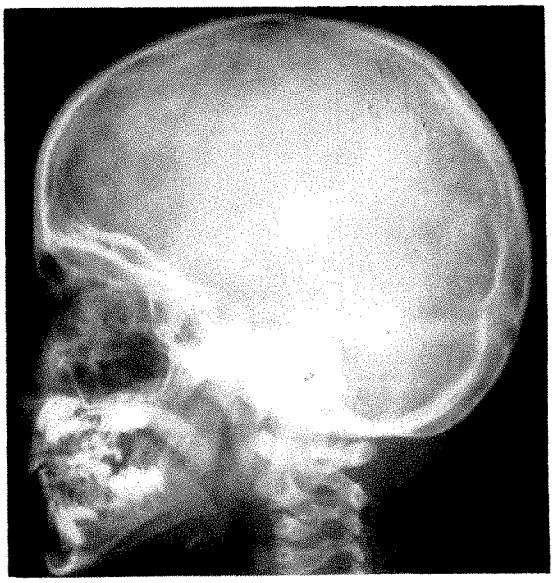


FIG. 1. Skull roentgenogram of a three and one-half year old female shows scattered foci of calcification, principally in the posterior part of the brain. The child had a mild microcephaly, with bilateral central choroiditis, and an I.Q. of 51.

* From the Departments of Radiology, Western Reserve University and University Hospitals of Cleveland.

DIFFERENTIAL DIAGNOSIS

Although cytomegalic inclusion disease is a much less common cause of widely distributed intracranial calcifications than is toxoplasmosis, a differential diagnosis between the two may be difficult. The dye test may not provide an immediate answer regarding toxoplasmosis, partly because it involves sending serum to one of the laboratory centers equipped to perform the test, but principally because a positive titer in a newborn infant is meaningless if the mother has been infected.

Cytomegalic inclusion disease may be equally difficult to prove if inclusion cells are not readily spotted in the excreta of the infant. The radiologist, therefore, may be asked whether there is anything about the lesions in the brain which will weigh in favor of one or the other diagnosis.

FINDINGS REPORTED IN THE LITERATURE

Several articles have suggested that a systematic arrangement of calcification lining the lateral ventricles may be helpful in favoring a diagnosis of cytomegalic inclusion disease. Mercer, Luse, and Guyton,[5] in their case report of the first patient to be diagnosed during life, state, "Ventriculograms confirmed impression that these areas of calcification were primarily in relation to ventricular walls."

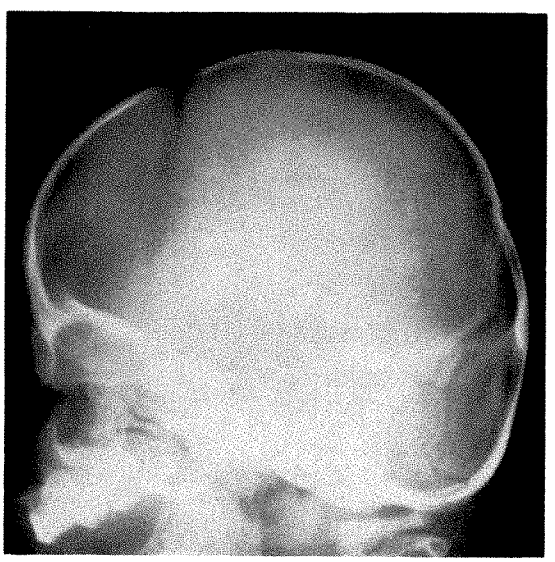


FIG. 2. Skull roentgenogram of a six month old female shows scattered foci of calcification in approximately a periventricular distribution, but air studies were not performed for exact localization. The clinical features were microcephaly, spastic seizures, and retinitis.

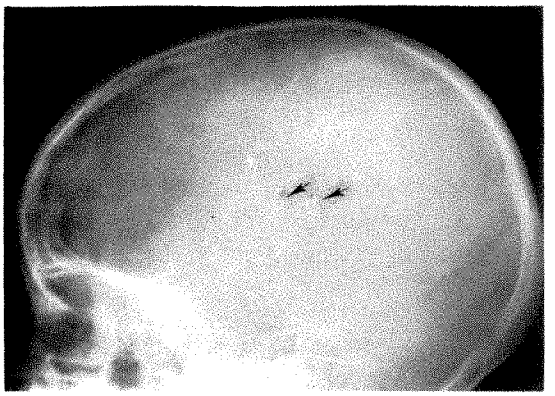


FIG. 3. A five and one-half year old male with a history of convulsions since the age of three. The patient was severely retarded, unable to talk or feed himself, and could walk only with assistance. Hearing and visual perception were markedly limited. He had the habit of hitting his head against the wall. The skull roentgenogram shows streaks of calcification (arrows) which are rather characteristic of toxoplasmosis.

Haymaker *et al.*[4] reported a case with advanced periventricular calcification, and concluded, "It seems likely that mental deficiency associated with periventricular calcification may in some instances be due to cytomegalic inclusion disease."

Sackett and Ford[6] showed extensive calcification of the linings of the ventricles in their case report. They apparently missed seeing the article by Haymaker *et al.* for they say, "Demonstration of the outline of the lateral ventricles of the brain on a RG of the skull is illustrated. This is the second such case reported, and in both the autopsy confirmed the impression of generalized cytomegalic inclusion disease."

Some recent articles are more emphatic. Daurelle, Smith, and Riemer,[2] in 1958, reported a case with calcium outlining the walls of the lateral ventricles, and suggested "that this radiographic finding, when pres-

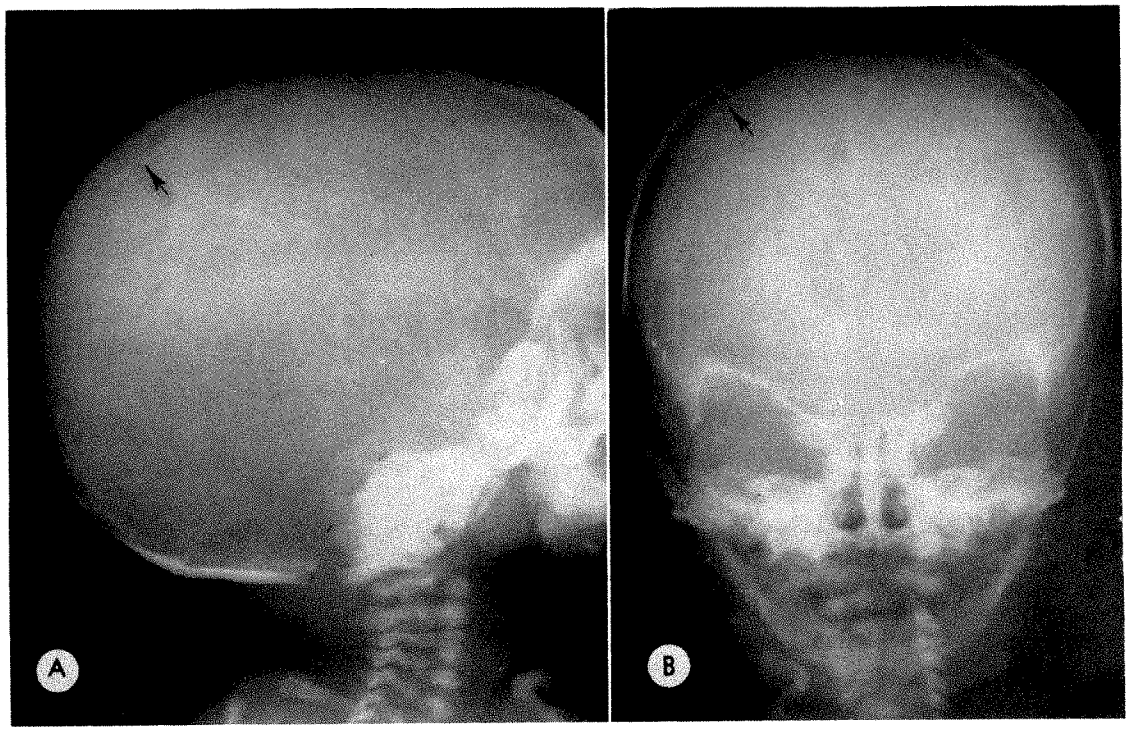
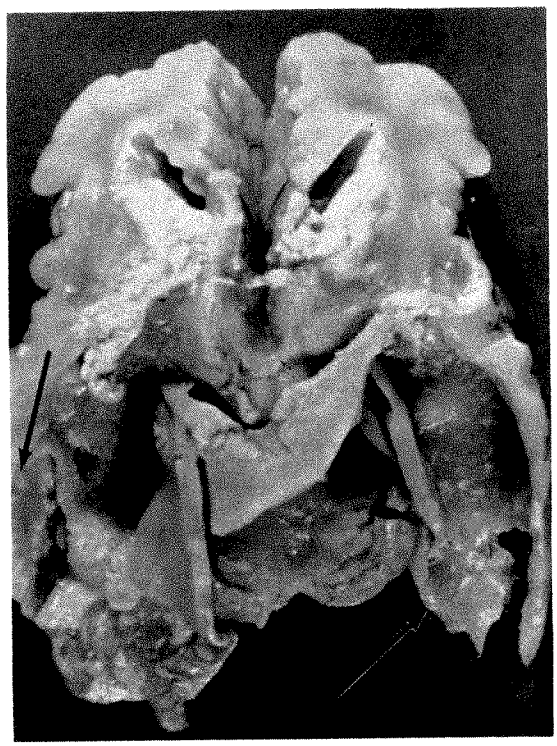


FIG. 4. (*A* and *B*) Calcification is seen in a distinct layer approximately 3 cm. within the calvarium and down the longitudinal fissure (arrows).



ent, indicates a diagnosis of cytomegalic inclusion disease, and may be pathognomonic of the disease."

Allen and Riley[1] at about the same time reported 2 cases, 1 of which showed calcification outlining dilated lateral ventricles, and they concluded, "Visible calcification outlining dilated lateral ventricles is probably pathognomonic when it occurs."

In the light of these experiences, we were presented with a case which exhibited extensive intracranial calcification.

REPORT OF A CASE

A premature male weighing 1,800 gm. was the product of a thirty-two week pregnancy. Routine physical examination at four hours had

FIG. 5. Formalin-fixed brain, sectioned longitudinally. The parietal and occipital cortex was severely thinned, and the posterior parts of the lateral ventricles were so dilated that their walls collapsed and infolded upon sectioning. Arrows point to flakes of calcium in the ependymal lining of the dilated ventricles.

revealed hepatosplenomegaly, and a blood smear showed a marked decrease in platelets. Upon admission at sixteen hours to Babies and Childrens Hospital there was, in addition, evidence of slight jaundice, and many petechiae scattered in clusters over the body. Coombs' test was negative. The jaundice rapidly became more severe. Exchange transfusion was started, during which the infant died at twenty-five hours of age.

Antemortem skull roentgenograms demonstrated calcifications of both hemispheres, principally in symmetric layers slightly within the cortex (Fig. 4, *A* and *B*). Autopsy revealed diffuse involvement of the organs by toxoplasmosis. Examination of the brain (Fig. 5 and 6) showed that the distribution of calcium was due to marked hydrocephalus, with lime deposited primarily in the ependymal lining.

CONCLUSION

On the basis of an autopsy diagnosis of toxoplasmosis of a newborn infant with a layer of calcium lining the dilated lateral ventricles, we are unable to substantiate any roentgenographic feature which can be considered to be pathognomonic of cytomegalic inclusion disease.

2065 Adelbert Road
Cleveland 6, Ohio

The author gratefully acknowledges the assistance of Dr. Robert P. Bolande in providing photographs from which Figures 5 and 6 were prepared.

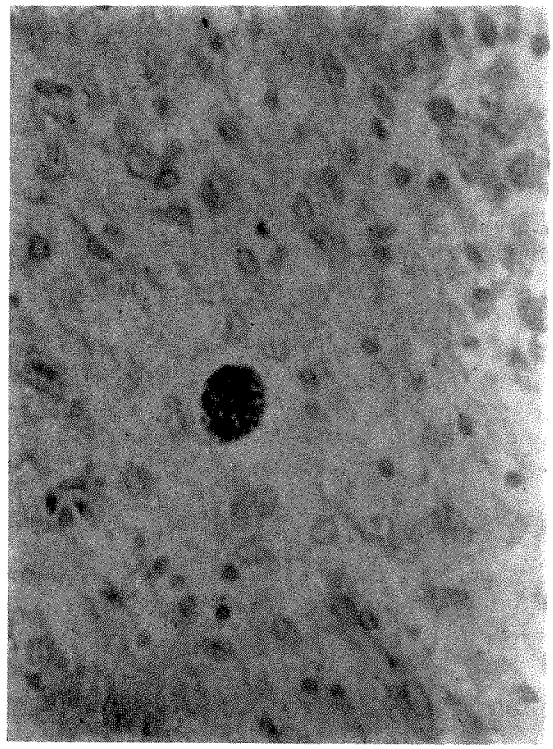


FIG. 6. Histologic section of the brain cortex of the patient shown in Figures 4 and 5. The *Toxoplasma* parasite is conspicuous.

REFERENCES

1. ALLEN, J. H., JR., and RILEY, H. D., JR. Generalized cytomegalic inclusion disease, with emphasis on roentgen diagnosis. *Radiology*, 1958, *71*, 257–262.
2. DAURELLE, G., SMITH, G. F., and RIEMER, W. Periventricular calcification and cytomegalic inclusion disease in newborn infant. *J.A.M.A.*, 1958, *167*, 989–991.
3. DYKE, C. G., WOLF, A., COWEN, D., PAIGE, B. H., and CAFFEY, J. Toxoplasmic encephalomyelitis; VIII. Significance of roentgenographic findings in diagnosis of infantile or congenital toxoplasmosis. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 830–841.
4. HAYMAKER, W., GIRDANY, B. R., STEPHENS, J., LILLIE, R. D., and FETTERMAN, G. H. Cerebral involvement with advanced periventricular calcification in generalized cytomegalic inclusion disease in newborn; clinicopathological report of case diagnosed during life. *J. Neuropath. & Exper. Neurol.*, 1954, *13*, 562–586.
5. MERCER, R. D., LUSE, S., and GUYTON, D. H. Clinical diagnosis of generalized cytomegalic inclusion disease. *Pediatrics*, 1953, *11*, 502–514.
6. SACKETT, G. L., JR., and FORD, M. M. Cytomegalic inclusion disease with calcification outlining cerebral ventricles. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *76*, 512–515.
7. WOLF, A., COWEN, D., and PAIGE, B. Human toxoplasmosis: occurrence in infants as encephalomyelitis verification by transmission to animals. *Science*, 1939, *89*, 226–227.

SPONDYLO-EPIPHYSEAL DYSPLASIA (PSEUDO-ACHONDROPLASTIC TYPE)*

By NEWELL FORD, M.D., FREDERIC N. SILVERMAN, M.D., *and* K. KOZLOWSKI, M.D.
CINCINNATI, OHIO

IN a classification of skeletal dystrophies, spondylo-epiphyseal dysplasias are said to comprise a subgroup of hereditary chondrodystrophies which is characterized by epiphyseal and vertebral modifications, of which the best known form is Morquio's disease.[2] As a rule, there would appear to be little opportunity for confusion between Morquio's disease and the well-known achondroplasia. In fact, the short trunk and relatively long extremities in cases of the former are considered to contrast with the relatively long trunk and short extremities in the latter condition.

Recently, Maroteaux and Lamy[3] reported observations in 3 dwarfed individuals who demonstrated features resembling achondroplasia but who, they believe, actually represented an extreme form of spondylo-epiphyseal dysplasia. The authors emphasize the fact that actual shortening of the extremities is frequent in Morquio's disease and can, at times, be so much accentuated as to cause confusion in diagnosis. The extreme form of spondylo-epiphyseal dysplasia, the pseudo-achondroplastic type, is demonstrated by the 3 patients who are the subjects of the present report.

REPORT OF CASES I AND II

CASE I. D.L.H., born September 17, 1936, was first seen at eight years of age in the Orthopedic Clinic at the Cincinnati General Hospital because of a painful left hip, dwarfism, and obesity. At birth, the patient weighed 6 pounds 4 ounces and was considered to be a normal full-term infant. She walked at ten months of age, talked normally and was an alert child. At approximately four to five years of age, she was observed to have a waddling gait and, at this time, it was also noted that she was appreciably shorter than other children of her age.

At the time of her first visit in the hospital, the patient was described as "dwarfed" with predominant shortening of the extremities and a relatively normal length of the trunk. She was obese and had an accentuated lumbar lordosis and scoliosis. She walked with a waddling gait. Her face and cranium were normal in appearance. The diagnosis of achondroplastic dwarfism was made. At the age of twelve years (Fig. 1, *A* and *B*), she was seen in the Endocrine Clinic at Children's Hospital because of the obesity and dwarfism. Shortly afterward, following mild trauma at an amusement park, she began to develop progressive quadriparesis and ultimately quadriplegia. Myelographic studies and roentgenographic examinations failed to demonstrate the local spinal complications of achondroplasia. After two cervical laminectomies and a period of convalescence, the patient stabilized with a paraplegia. Because of her normal intelligence, she was able to complete high school in spite of the neurologic handicaps mentioned. Various laboratory studies were normal including calcium, phosphorus, alkaline phosphatase, basal metabolic rate and radioactive iodine uptake. At the age of sixteen, she weighed 77 pounds and measured 39 inches in height. She is now twenty-four years of age and confined to a wheelchair.

CASE II. S.H., born May 1, 1934, was seen in the Endocrine Clinic of the Children's Hospital while accompanying his sister, Case 1, to the clinic. He had been born at term as an apparently normal infant and his early development was reported as normal. His short stature and waddling gait were not noted until he was between four and five years of age. At the age of fourteen (Fig. 2, *A* and *B*), he weighed $45\frac{1}{2}$ pounds and was $36\frac{3}{4}$ inches in height. He was of average intelligence and subsequently was able to complete high school without difficulty. Be-

*From the Departments of Pediatrics and Radiology, College of Medicine, University of Cincinnati; the Children's Hospital and Children's Hospital Research Foundation; and the X-Ray Department of the First Pediatric Clinic of the Medical School of Poznan, Poland.

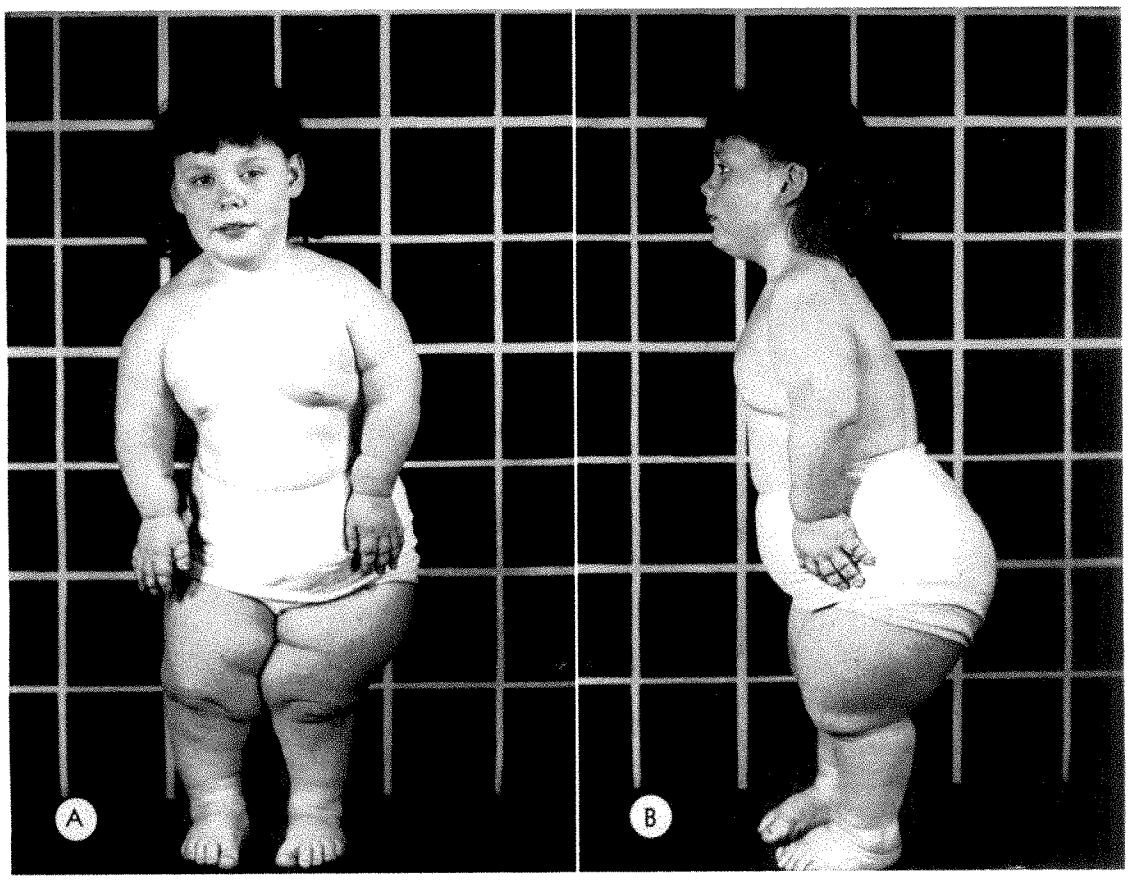


Fig. 1. Case 1. (*A* and *B*) Photographs at twelve years of age. Because of the obesity, the face may seem to have some mild achondroplastic characteristics. However, note that the features are delicate and that a true saddle nose deformity and protruding brow are not present.

cause there were no complications to his dwarfism, this patient was not examined as carefully as his sister, although we were able to obtain clinical photographs at the time of his visit and roentgenographic examinations as well. It was thought that he and his sister both suffered from achondroplasia.

FAMILY HISTORY

The mother of these 2 patients was of normal height and weight and came from a family in which dwarfism or other abnormalities of stature were denied. The father had been a "dwarf in a circus," measuring 4 feet 9 inches in height. He apparently was also a chronic alcoholic and died from the complications of this condition several years after the children were first seen. He had never been seen by a member of the hospital departments. Consanguinity was denied. Information concerning the father's antecedents and collaterals is incomplete, but the children's mother stated that she did not believe there were other affected individuals in the father's family. A pedigree, as developed from conversation with the mother, is diagrammed in Figure 3.

ROENTGENOGRAPHIC FINDINGS

Skull (Fig. 4, *A* and *B*). In both children, the skull was normal in appearance and showed no evidence of brachycephaly, frontal protuberance or bossing, or of saddle nose deformity. Abnormal shortening of the cranial base was not demonstrated. Developmental features were commensurate with the age.

Extremities (Fig. 5, *A* and *B*; 6, *A* and *B*). Severe deviations from normal were observed in all the metaphyses and epiphyses of the tubular bones including the bones of the hands. The metaphyseal areas were enlarged, irregular, and had a mushroomed appearance. The epiphyses were small, irregular, fragmented,

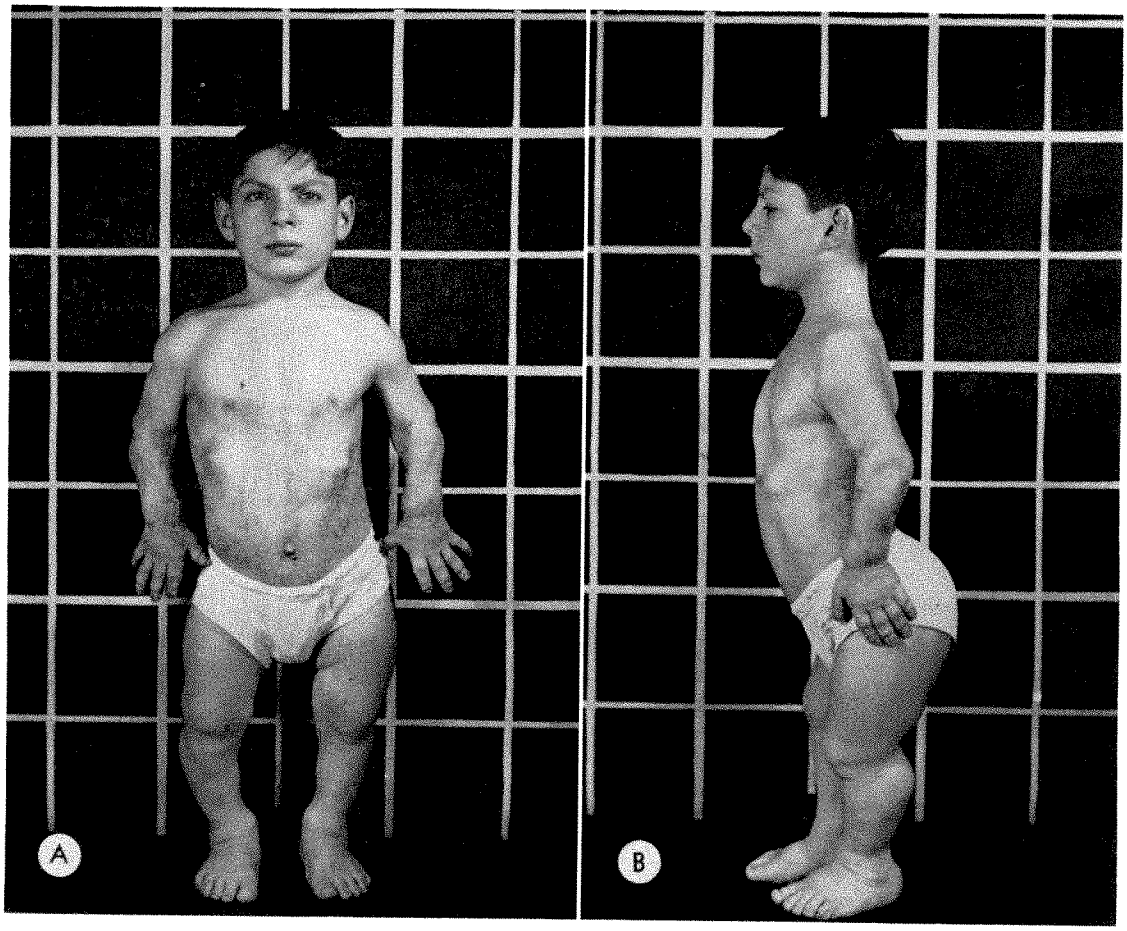


FIG. 2. Case II. (*A* and *B*) Photographs at fourteen years of age. The normal head and face are better shown on these photographs than on those of the sister (Fig. 1, *A* and *B*), although the achondroplastic proportions are prominent.

irregularly ossified and deformed. The diaphyses were shortened and tended to bow outward. The phalanges were short, wide, "stubby" and had irregular wide metaphyses. The cortices were not abnormally thickened, although the bones were relatively thick for their length.

Spine and Ribs (Fig. 7, *A*, *B* and *C*). Both patients demonstrated a rather prominent scoliosis, more marked in the girl than in the boy. The vertebral bodies were somewhat flattened and had a tendency to a biconvex form. On the lateral roentgenograms, some anterior "beaking" was visualized, particularly in the lumbar vertebrae. The ribs had a spatulate appearance produced by an increased breadth lateral to relatively normal necks. The ribs were somewhat short and their sternal ends were cupped and enlarged.

Pelvis (Fig. 8). The pelvis was small in both patients. The greater sciatic notch was not markedly reduced in size. Irregular mineralization of the femoral heads and necks and the adjacent acetabula was a very prominent feature.

REPORT OF CASE III

CASE III. A nine year old girl was admitted to the First Pediatric Clinic in Poznan, Poland, December 1, 1959, because of short stature, deformities of the upper and lower limbs, and a waddling gait. The admission diagnosis was achondroplasia. The patient was the result of the tenth pregnancy of healthy parents. The mother was thirty-seven and the father fifty-four years of age when the child was born. Only 2 brothers, results of the first two pregnancies, were living, aged twenty and twenty-

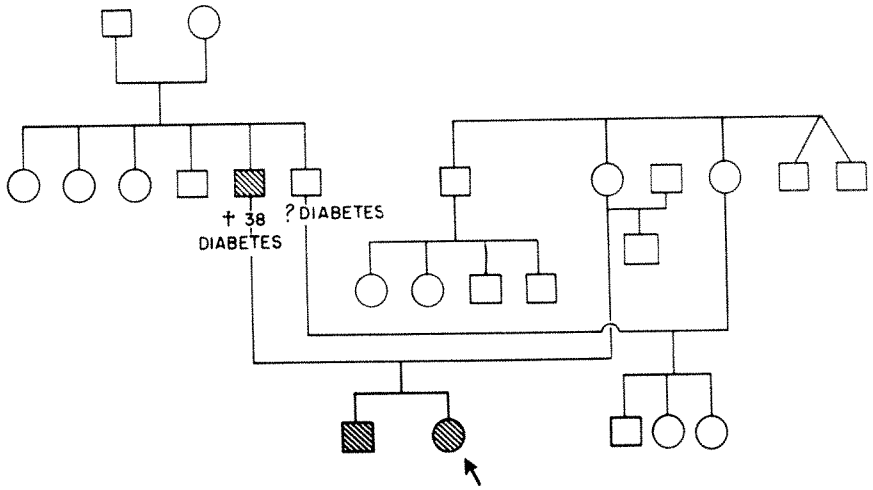


Fig. 3. Case I and II. Diagram showing pedigree which was developed from the information obtained from the patients' mother; none of the other members of the family have been examined.

two years, respectively. In the interval, there had been 6 miscarriages induced by quinine and 1 artificial premature labor. During the second month of the pregnancy with the patient, the mother ingested considerable quinine in an unsuccessful attempt to induce abortion. The amount ingested was apparently sufficient to cause intoxication.

The child weighed 3,300 gm. at birth. The length was not recorded but the child was said to be normal. She was breast-fed until the fourth month of life and subsequently was given an adequate diet. Vitamin D-2 (600,000 units) was administered on three occasions in the first year of life.

At seven months of age the mother was informed that the child was short; she had not observed any abnormality in size up to this time. Some weeks later, a slight bowleg deformity was noted. Motor development was slightly retarded. The patient sat at the age of seven months, stood up at one and one-half years and walked at three years. The mental development, however, had been normal. Talking began at eighteen months and the child was able to read and write to a degree commensurate with her age.

At the age of eight years, the diagnosis of achondroplasia was made when the child was hospitalized because of the short stature. The parents, brothers and other members of the family were of normal stature and there was no

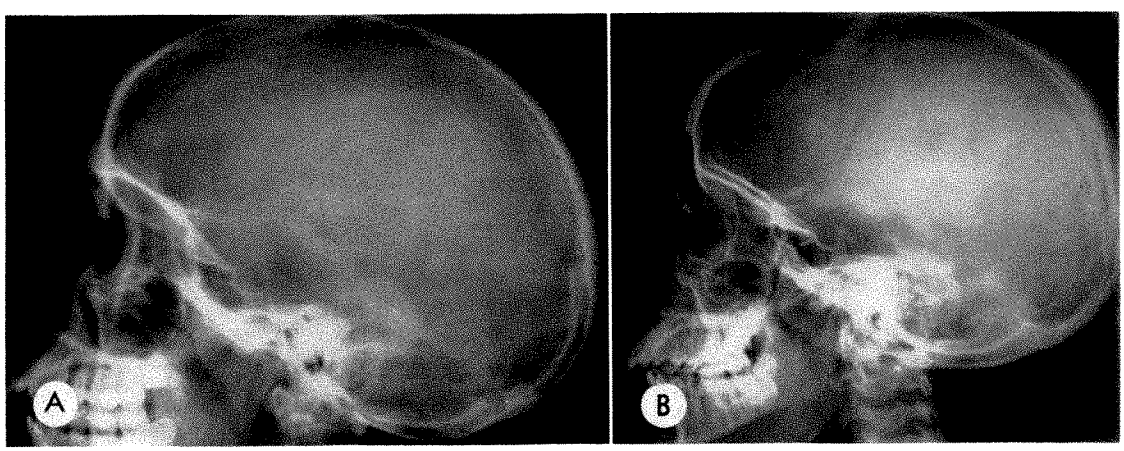


Fig. 4. Lateral skull roentgenograms of (*A*) Case I and (*B*) Case II are normal and show no evidence of achondroplasia.

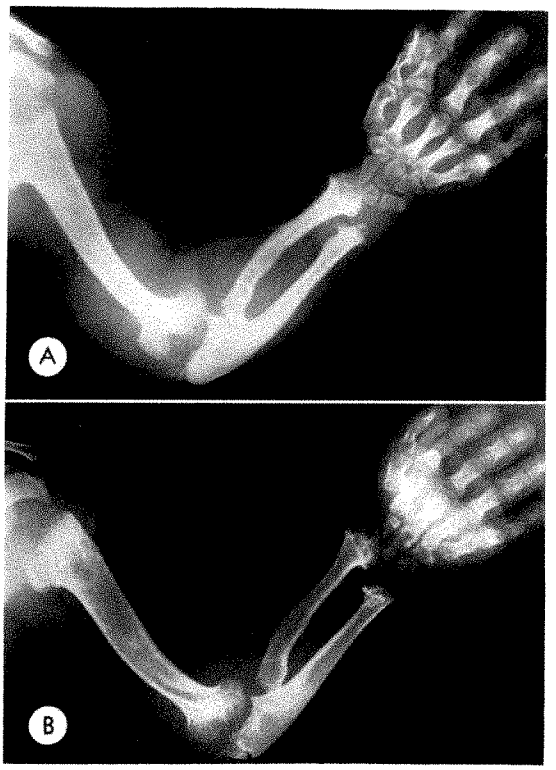


FIG. 5. Roentgenograms of the upper extremities of (*A*) Case I and (*B*) Case II show short and thick bones, excessive flaring of the metaphyses, and marked irregularity and fragmentation in the unfused ossification centers.

history of skeletal disease.

On physical examination, the child was found to be well nourished but short in stature (height —92 cm., weight—17 kg.) (Fig. 9, *A*, *B* and *C*). The upper and lower limbs were short in relation to the length of the trunk and the head. An exaggerated lumbar lordosis was present. The lower extremities were bowed and a waddling gait was present. Marked enlargement of the joints was noted. The motions of the large joints were limited. Muscle tone, volume and strength were normal. The head was normal. Examination of the ears, nose and throat revealed moderate hypertrophy of the tonsils and adenoids. The teeth were normal. The clavicles were overlong without exaggeration of the curves; some flaring of the lower thoracic cage was noted. Measurements of the upper limbs are shown in Table I.

Laboratory findings revealed a normal red blood cell count, white blood cell count and hemoglobin; urinalysis (specific gravity 1.025,

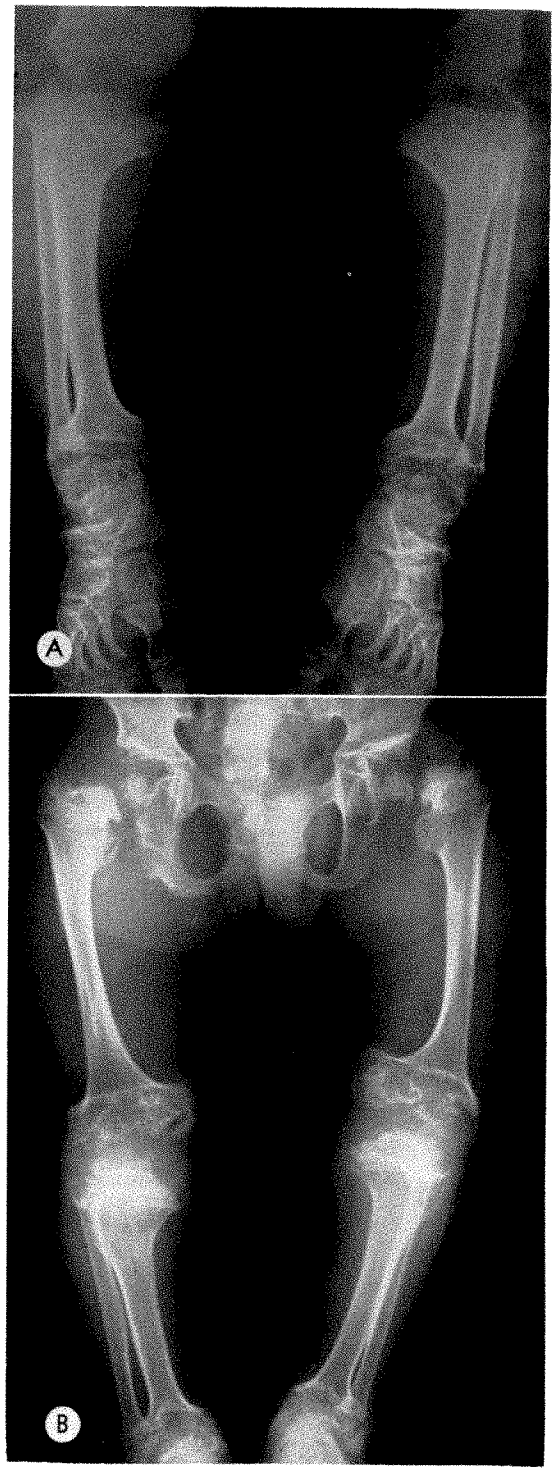


FIG. 6. Roentgenograms of lower extremities of (*A*) Case I and (*B*) Case II show the extremely irregular fragmented appearance of the ossification centers. The cortices are not abnormally thick.

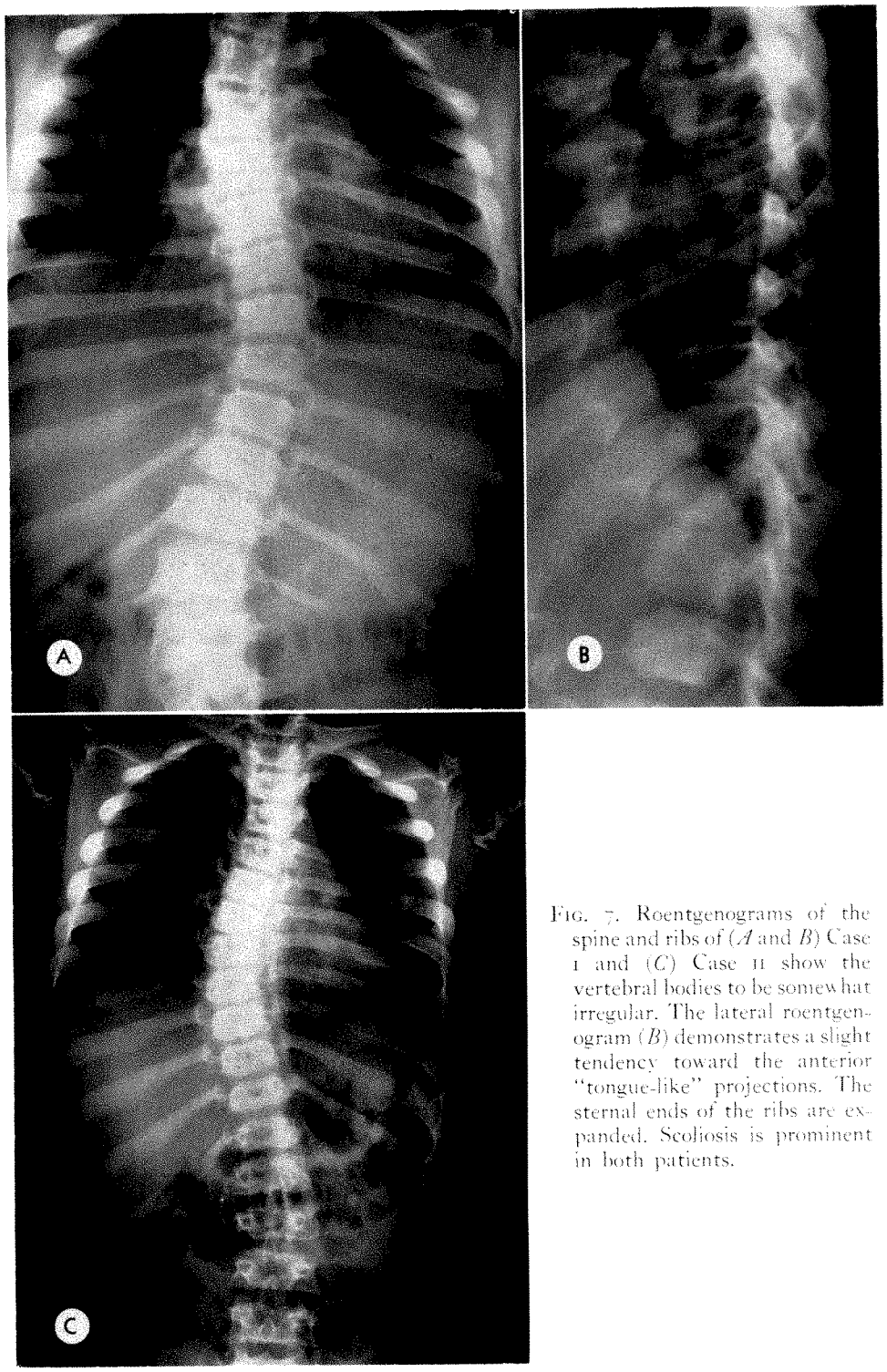


FIG. 7. Roentgenograms of the spine and ribs of (*A* and *B*) Case I and (*C*) Case II show the vertebral bodies to be somewhat irregular. The lateral roentgenogram (*B*) demonstrates a slight tendency toward the anterior "tongue-like" projections. The sternal ends of the ribs are expanded. Scoliosis is prominent in both patients.

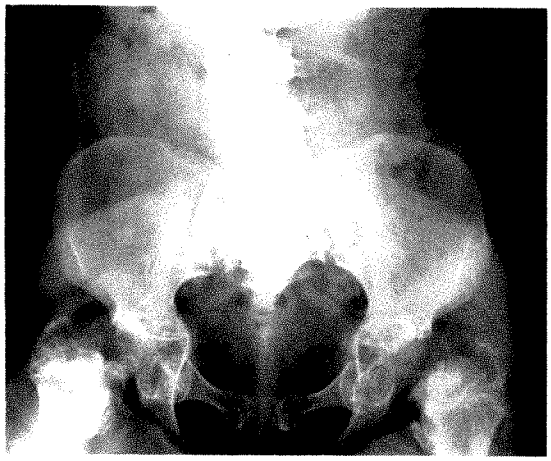


FIG. 8. Case I. Roentgenogram of the pelvis shows only slight diminution in the length of the sacroiliac notch notwithstanding the marked lordosis of this patient. See also Figure *6B*. Neither pelvis is that of classic achondroplasia.

TABLE I

MEASUREMENTS OF CASE III

	Length (cm.)	Normal Length* (cm.)
Upper Limb (acromion to third finger)	34	57
Forearm and Hand (radial point to third finger)	19	33
Hand (styloid point to third finger)	11	14
Sitting Height	66	66
Lower Extremities (anterior superior iliac spine to ground)	41	71
Leg (tibial point to ground)	20	34
Foot (malleolus to ground)	5	5

* These figures, corresponding to normal lengths for a nine year old female, were provided by Dr. Pierre Maroteaux.

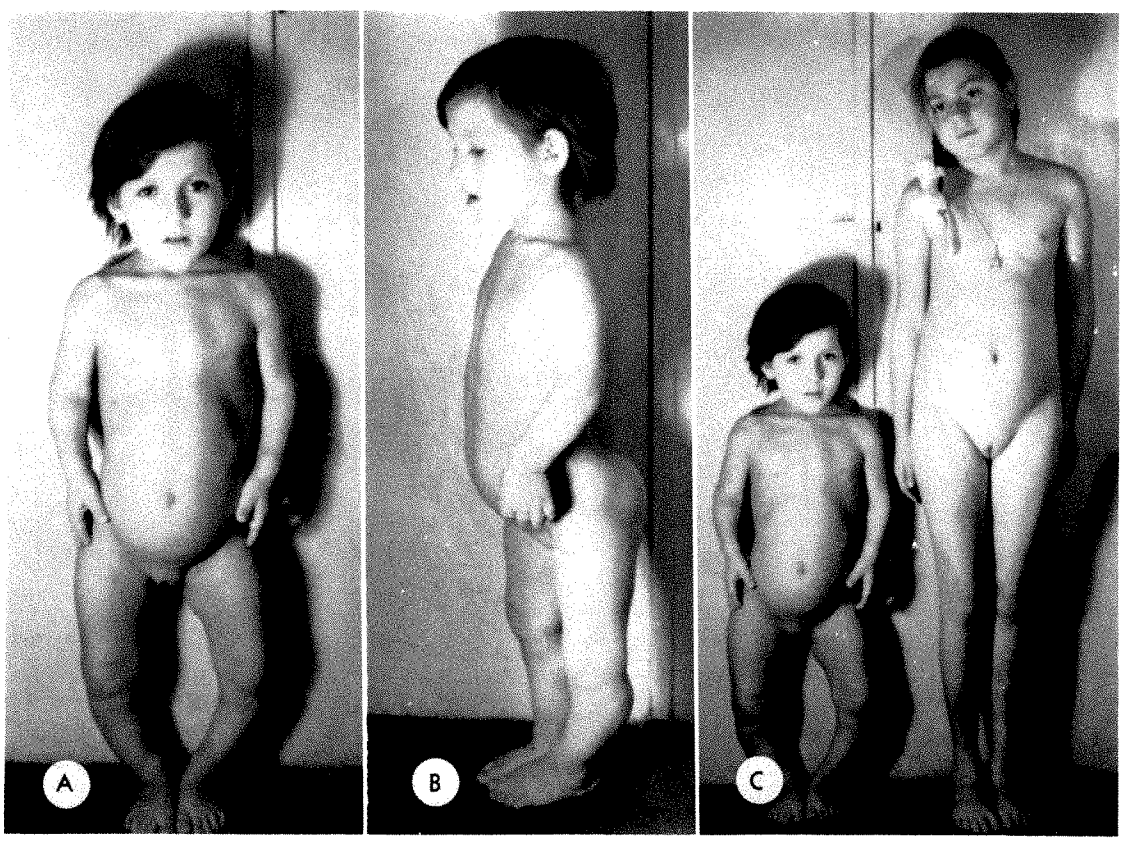


FIG. 9. Case III. Photographs at nine years of age. (*A*) Frontal view. (*B*) Lateral oblique view. (*C*) Comparison with normal female of nine years.

pH 6.9); normal blood and urine chromatography and normal excretion of calcium, phosphate and ammonia in a twenty-four hour urine specimen. The serum calcium was 10.4 mg. per cent; phosphorus 4.3 mg. per cent; serum alkaline phosphatase 11.3 Bodansky units; nonprotein nitrogen 21 mg. per cent; serum urea 30 mg. per cent; creatine 1.2 mg. per cent; cholesterol 176 mg. per cent. The serum sodium, potassium and chloride concentrations were normal. The glucose tolerance test was normal; 7 per cent fat in stool; trypsin test was positive in 1:5 and 1:10 dilutions, negative in 1:50 and 1:100. Electrophoretic protein pattern: total protein 6.2 gm. per cent, albumin 60 per cent, alpha$_1$ 4.2 per cent, alpha$_2$ 7.5 per cent, beta 9.3 per cent, and gamma 19 per cent.

An excision biopsy of the costochondral junction of the left sixth rib was performed. Only the cartilaginous portion was taken. This appeared to be grossly normal; upon section, the cells in the cartilage were noted to be irregularly disposed. Beneath the perichondrium, the cells were larger than in the deeper layers. Some free spaces were seen around the cells. The bone substance of the cartilage was swollen in several areas. In the deepest layers of the cartilage, the cells were gathered in groups of less than 20. Under the perichondrium, some small collections of capillaries were seen. No tissue characteristic of achondroplasia was noted and no connective tissue was observed at the cartilage-shaft junction.

ROENTGENOGRAPHIC FINGINGS

Skull (Fig. 10). The roentgen appearance of the skull and teeth was normal. The foramen magnum was normal in size.

Extremities (Fig. 11, *A*, *B* and *C;* and 12). Roentgen examination of the bones of the upper and lower extremities (Fig. 11, *A*, *B* and *C*) demonstrated these structures to be short and thick with otherwise normal cortex. A moderate varus deformity was present at the knees. All the normal curves of the long bones were exaggerated. The ends of the shafts were flaring, irregular and fragmented. Skeletal age of the hands and wrists was approximately five years according to the standards of Greulich and Pyle (Fig. 12). The component bones were abnormally short and thick and demonstrated irregularities in the epiphyseal and metaphyseal areas comparable to those noted in the large tubular bones.



Fig. 10. Case III. Roentgenogram shows normal skull. The lack of cranial and facial involvement is an important differential point with respect to achondroplasia.

Spine and Ribs. In the spine there was a prominent lordosis. Lumbarization of the first sacral segment was present. The upper and lower borders of the lumbar vertebral bodies were irregular but no other abnormalities were found. In particular, no marked flattening of the vertebral bodies and no anterior tongue-like projections were present. On the antero-posterior roentgenogram, no distinct scoliosis was demonstrated. The scapulae were not remarkable. The ribs were of normal length and shape but irregularity of the zones of provisional calcification was noted at the costochondral junctions. The clavicles were long but the curvature was normal.

Pelvis (Fig. 13). In the pelvis the iliac bones were somewhat shortened below the posterior inferior iliac spine. The greater sciatic notches were reduced in size. The acetabular angles were flattened. The sacrum was normal in shape and the ischiopubic synchondroses were open.

DISCUSSION

The chief features, described by Maroteaux and Lamy,[3] in differentiating the pseudo-achondroplastic form of spondylo-epiphyseal dysplasia from classic achondroplasia are clearly demonstrated by these 3 cases. These include: (1) the late date of discovery of the abnormality; (2) the ab-

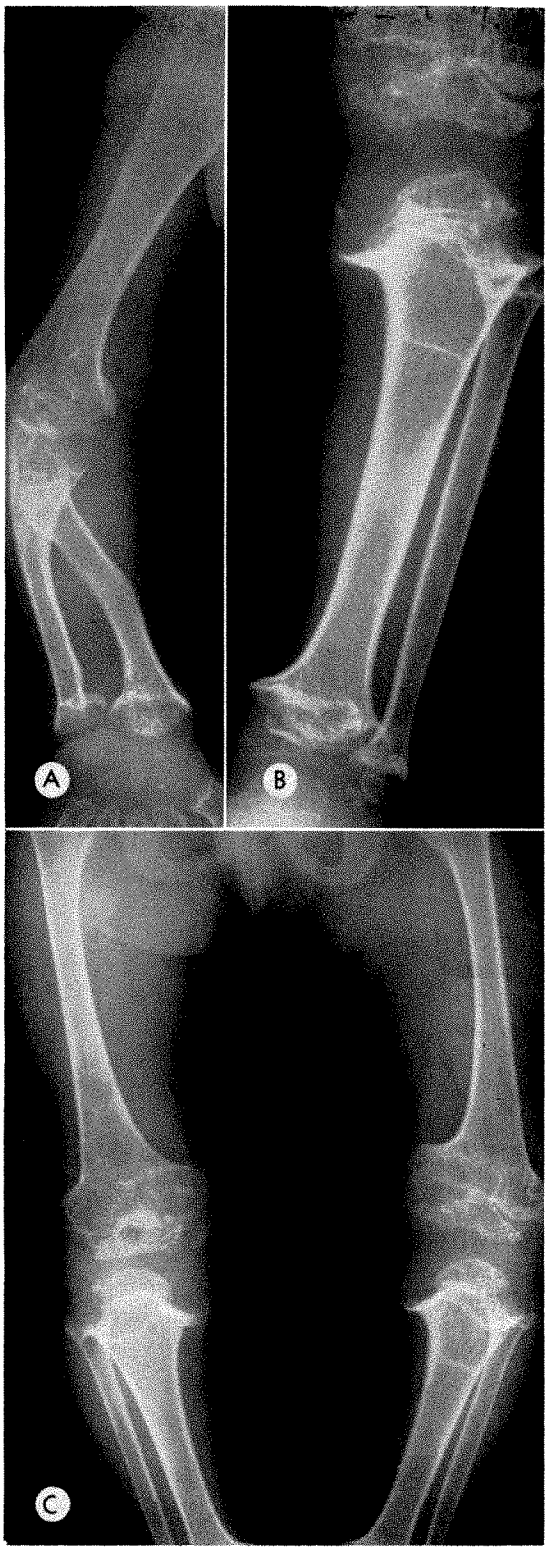


FIG. 11. Case III. Roentgenograms of the (*A*) upper extremity, (*B*) lower extremity and (*C*) knees show the flaring of the metaphyses, the marked irregularity of the epiphyses and the normal cortex.

sence of clinical and roentgenologic abnormalities in the face and skull; and (3) the roentgenologic findings of gross irregularities in the epiphyseal-metaphyseal areas with fragmentation of the epiphyseal ossification centers. Our first 2 patients demonstrated abnormalities in the vertebral column which were not a prominent finding in the third patient. An additional point worthy of note is the relative length of the sacroiliac notch in our patients as well as in those described by Maroteaux and Lamy.[3] This contrasts significantly with the small sacroiliac notch which has been emphasized recently by Caffey[1] as a pelvic manifestation of classic achondroplasia.

It is unfortunate that actual measurements in the newborn period are not available of any of the patients reported thus far. The delay in recognition of the disease is considered an important differential feature but is dependent exclusively on nonobjective observations by the parents. It is well known that mild or even moderate achondroplastic dwarfs may not be recognized at birth unless a clue is provided by roentgenologic examination; nevertheless, dwarfism as marked as that noted in our patients would be expected to have been recognized much earlier in life than at the end of the first year (Case III) or the third to fifth year (Case I and II) if it were caused by classic achondroplasia.

The obvious escape of involvement of the cranial structures would appear to be an extremely important differential point. Spinal complications in classic achondroplasia have been documented by Vogl and Osborne[7] and by Spillane.[6] The occurrence of signs of transverse myelopathy in our patient (Case I) was originally thought to support further the diagnosis of achondroplasia. Obviously, the complication, if a result of diminished intraspinal space, should be expected to occur in any condition in which the space is compromised, including

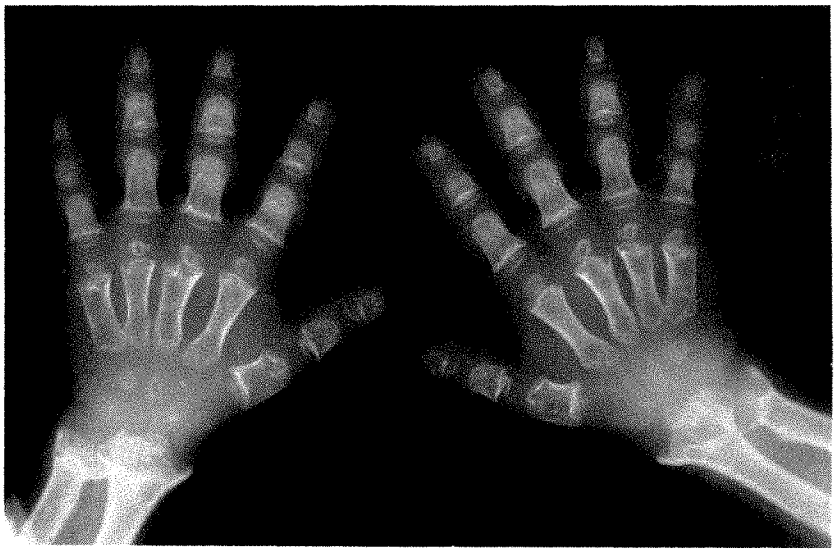


Fig. 12. Case III. Roentgenogram of the hands and wrists shows short stubby bones, irregularity in epiphyseal ossification centers and poor development of carpal ossification centers.

the pseudo-achondroplastic form of spondylo-epiphyseal dysplasia.

The roentgenologic features of spondylo-epiphyseal dysplasia certainly differ from those of classic achondroplasia, but experienced observers had long noted incomplete and intermediate forms which suggest transitional forms of cartilaginous dystrophies. With the present interest in enzymatic defects as the basis for disease processes, it is tempting to consider the various forms of this group of skeletal dystrophies as resulting from the various combinations of certain enzymatic processes which affect cartilage proliferation, susceptibility of cartilage matrix to calcification, and transformation of calcified cartilage to bone. If we also include factors which determine anatomic distribution and time and sequence of action[5] of the various defects, we are provided with a concept which brings together these various conditions. This, of course, takes us directly into the field of genetics, and here the studies of heredity of the several diseases may provide additional information. However, it is pertinent to point out that there are difficulties in differentiating the mutational origin of a dominant condition from the unveiling of a recessive characteristic. Moreover, it is quite possible that phenocopies (*i.e.*, non-genetic simulations of genetically determined traits) may occur in this group of malformations as well as in others. In our Case III, the mother was attempting abortion during an important developmental period. The clinical and roentgenologic findings, although similar to those of the first 2 cases and to those described by Maroteaux and Lamy, differ to some extent in the relatively unaffected spine, and

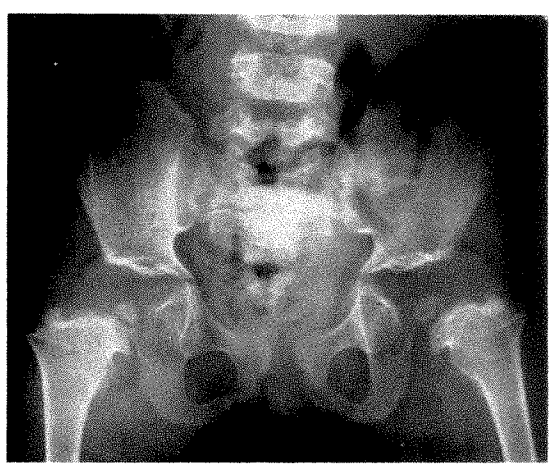


Fig. 13. Case III. Roentgenogram of the pelvis shows the marked irregularity of the epiphyseal ossification centers. The sacroiliac notch is somewhat shorter than in Cases I and II and approaches more closely the findings in achondroplasia.

the pelvis in which resembles more that of the true achondroplast. Furthermore, there is no evidence of any hereditary disease in the antecedents.

There can be little doubt of the dominant nature of disease in Cases I and II. This heredity differs from the recessive nature of the disease described by Morquio and which bears his name. In 1957, Maroteaux, Lamy and Bernard[4] reported a form of spondylo-epiphyseal dysplasia with a late onset (second decade) which they believe is transmitted by a recessive sex-linked gene. These individuals had clinical and roentgenologic findings closely resembling those of Morquio's disease. It would therefore appear that the various approaches to classification—clinical manifestations, evolution, roentgenologic features and genetic nature—are all collectively inadequate and individually misleading until a basic defect, probably biochemical, can be found. Meanwhile, however, it would appear that the description of the variants and the grouping of these variants with respect to their common features may point toward the area in which a basic defect should be sought.

SUMMARY

Three patients with a pseudo-achondroplastic form of spondylo-epiphyseal dysplasia are described. The findings conform very closely to the condition as originally reported by Maroteaux and Lamy.[3] Each patient was first considered to have classic achondroplasia. The features which differentiate spondylo-epiphyseal dysplasia from classic achondroplasia and which were present in our patients include: (1) delayed onset of dwarfism, (2) lack of involvement of the head and face, and (3) roentgenographic irregularities in the epiphyses. A fourth finding with respect to the shape of the pelvis in achondroplasia, as described by Caffey, is added to the differentiating signs.

Frederic N. Silverman, M.D.
Children's Hospital
Cincinnati 29, Ohio

The authors wish to thank Dr. Josef Warkany for permission to publish Cases I and II, and Dr. Pierre Maroteaux for his advice and kindness in reviewing our material.

REFERENCES

1. CAFFEY, J. Achondroplasia of pelvis and lumbosacral spine; some roentgenographic features. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *80*, 449–457.
2. LAMY, M., and MAROTEAUX, P. Les chondrodystrophies génotypiques. L'Expansion Scientifique Française, Paris, 1960, pp. 9–15.
3. MAROTEAUX, P., and LAMY, M. Les formes pseudo-achondroplastiques des dysplasies spondylo-épiphysaires. *Presse méd.*, 1959, *67*, 383–386.
4. MAROTEAUX, P., LAMY, M., and BERNARD, J. La dysplasie spondylo-épiphysaire tardive; description clinique et radiologique. *Presse méd.*, 1957, *65*, 1205–1208.
5. RUBIN, P. Personal communication.
6. SPILLANE, J. D. Three cases of achondroplasia with neurological complications. *J. Neurol., Neurosurg. & Psychiat.*, 1952, *15*, 246–252.
7. VOGL, A., and OSBORNE, R. L. Lesions of spinal cord (transverse myelopathy) in achondroplasia. *Arch. Neurol. & Psychiat.*, 1949, *61*, 644–662.

Vol. 86, No. 3

GARGOYLISM: A REVIEW OF THE ROENTGEN SKULL CHANGES WITH A DESCRIPTION OF A NEW FINDING*

By W. DONALD HORRIGAN, M.D., *and* DAVID H. BAKER, M.D.
NEW YORK, NEW YORK

GARGOYLISM (Hurler's syndrome), a metabolic disorder characterized by the overproduction of certain mucopolysaccharides which accumulate in connective tissue as well as in the cells of many organs, gives rise to a distinct clinical syndrome. This entity was first recognized by Hunter[7] in 1917 and then was described in greater detail by Hurler[8] in 1920. Since that time numerous reports[3–6,9–11] have appeared in the medical literature, describing the varied aspects of this rare disorder.

The clinical syndrome is characterized by dwarfism, infantilism, mental retardation, coarse facial features, large tongue, pot belly, hepatomegaly, splenomegaly, kyphosis, corneal clouding and characteristic skeletal dystrophy. Overproduction of certain normal polysaccharides results in their accumulation in most organ systems including the nervous, cardiovascular, reticuloendothelial, endocrine, and skeletal systems and explains the protean manifestations of the disease. The excess production of these polysaccharides is accompanied by the excretion of large quantities in the urine which provides the basis for a quantitative test.[12]

Although the majority of infants with this disorder are normal at birth and develop the characteristic clinical picture toward the end of the first year of life,[14] Caffey has described the roentgen appearance of prenatal and neonatal bone lesions and their subsequent postnatal evolution.[1] When all of the stigmata are present, the disease is readily recognized, but variants, with one or more major features being absent, do occur. It is this latter group which has been confused with Morquio's disease, achondroplasia, and hypothyroidism. From the evidence currently at hand, it would appear that gargoylism is genetic in origin with at least two distinct genotypes—a sex-linked recessive and an autosomal recessive form.[12]

The roentgenologic appearance of the skeleton in gargoylism, although somewhat similar to that seen in Morquio's disease, is distinctive and a prominent feature of the clinical syndrome. In the extremities, the changes, which are more pronounced in the upper limbs, consist of swelling of the diaphyses with irregular thickening of the cortex. The ends of the bones are tapered and in the humerus the surgical neck is angled dorsad. The distal epiphyseal plates of the radius and ulna usually face toward each other. The changes in the hands consist of pointing of the base of the metacarpal bones and a "bullet-nose" appearance of the phalanges. In the lower extremities, the changes are less marked, but usually coxa valga, narrowing of the proximal one-third of the femur and poor mineralization of the femoral head are noted. A kyphotic deformity of the spine at the thoracolumbar level is due to irregular growth and deficiency of the superior half of the vertebral bodies. This is occasionally associated with slipping and a gibbus deformity.

The skull is unusual in configuration with a circumference that is always greater than normal. The dolichocephalic type of skull predominates although other types have been noted. Prominence of the frontal bones with heavy supraorbital

* From the Departments of Pediatrics and Radiology, College of Physicians and Surgeons, Columbia University and the Babies Hospital, New York, New York.

ridges is a common finding. The sella turcica is elongated and shallow with a long anterior recess extending beneath the anterior clinoid processes—the so-called "J" shaped sella. The mandible is frequently broad and heavy with the teeth widely spaced, irregular and poorly developed. In addition, we have recently observed an abnormality of the mandibular condyle which appears to be specific for gargoylism and has prompted us to review the skull roentgenograms in this unusual disorder.

MATERIAL AND RESULTS

The skull roentgenograms of the last 20 patients with gargoylism seen at the Babies Hospital were reviewed to determine the specificity of the condylar change to be described and to document the nature of the usual skull changes associated with this syndrome. The age of the patients at the time of examination ranged from two months to twelve years with the majority being in the two to six year age group. There were 13 males and 7 females. All patients fulfilled the criteria of gargoylism on clinical and roentgenologic grounds and those tested were found to excrete excessive quantities of chondroitin and heparitin sulfate in the urine. The quantity excreted did not correlate with the severity of the clinical or roentgenologic manifestations. The skeletal changes ranged from minimal to the classic far advanced changes.

All types of skull configurations were encountered in this group, but the most common was the dolichocephalic type (Fig. 1, *A* and *B*). Prominent interdigitations along the coronal suture were observed frequently and appeared to be related to the age of the patient at the time of examination. With the exception of one patient who was eight years old, all examples of prominent interdigitations were noted in the two to three year age group (Fig. 2).

The sella turcica in all instances was elongated and shallow with a long anterior recess extending beneath the anterior clinoid processes (Fig. 3). This is the so-called "J" shaped sella which is nonspecific and can occur as a normal variant as well as in other disease states.

Prominent supraorbital ridges with associated depression of the bridge of the nose were noted in 8 patients. The orbits in all

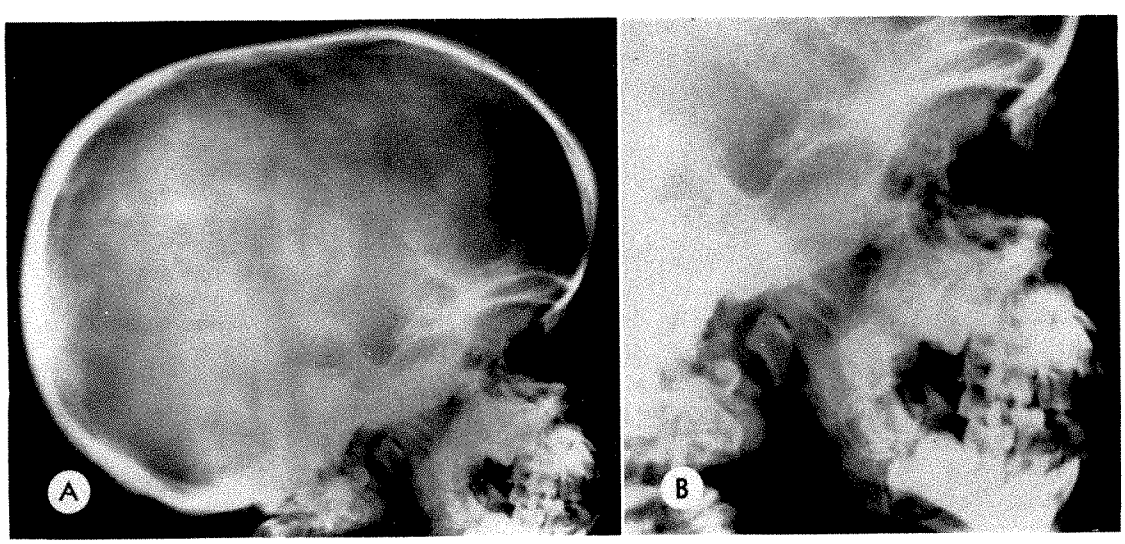


FIG. 1. (*A*) U91547-4 Four year old white male with typical clinical and roentgenographic features of gargoylism. The lateral roentgenogram of the skull demonstrates the long anteroposterior dimension, the "J" shaped sella turcica and the flat, slightly concave condylar surface of the mandible. (*B*) The flattened and slightly concave articular surface of the mandibular condyle is demonstrated to better advantage.

patients studied were of normal configuration. Special studies of the optic foramina were not available for review. Sinus development was markedly retarded with absence of the frontal sinus noted in every patient. Likewise, mastoid development was poor and lack of pneumatization of the sphenoid sinus was a constant finding.

The mandible presented a rather characteristic appearance on the frontal roentgenogram (Fig. 4). It was short and broad with a resultant square configuration. This appearance was found in 75 per cent of the patients studied. Dentition was also abnormal with the teeth irregularly spaced and poorly developed. Despite these abnormalities, no complaints referable to difficulty in mastication were noted.

The condyle of the mandible was observed to have either of two characteristic appearances. In the patients with marked skeletal changes, the condyle had a concave superior surface (Fig. 1, *A* and *B*, 3, and 5) while in those patients with less marked skeletal disease, the condyle presented a flattened articular surface (Fig. 6). This was noted in 12 of 15 patients where the condyles could be satisfactorily visualized on the lateral skull roentgenograms. In order to determine whether the abnormal appearance of the mandibular condyle was merely fortuitous, the skull roentgenograms of a group of patients with achondroplasia, Morquio's disease, and abnormal skull configurations were reviewed. In no instance was a similar appearance of the mandibular condyle noted. Thus, this condylar configuration may be specific for gargoylism.

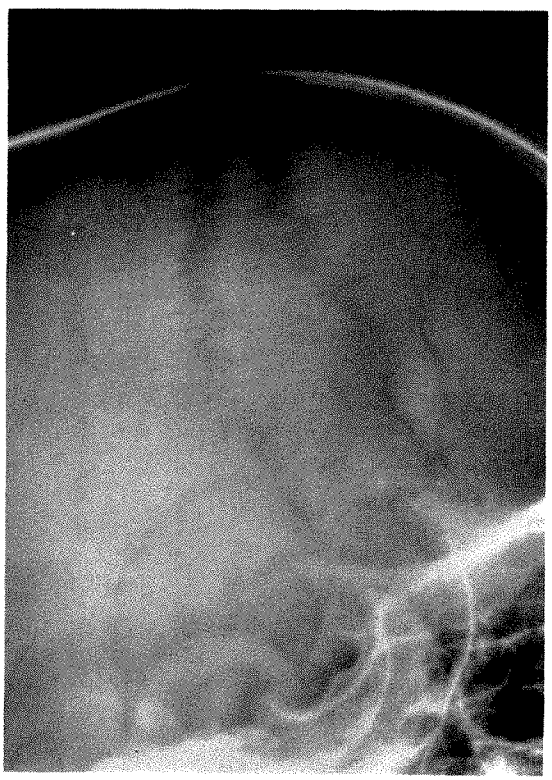


FIG. 2. U1376758 Two year old Negro male with typical features of gargoylism. Note the prominent interdigital markings along the coronal suture as well as the elongated sella turcica.



FIG. 3. U1368447 Four year old white female with typical skeletal features of gargoylism and positive urinary chondroitin test. The lateral roentgenogram of the skull shows the elongated "J" shaped sella turcica and the concave superior articular surface of the mandibular condyle.

DISCUSSION

The condylar abnormality in gargoylism has been alluded to previously in the literature, but to our knowledge has never been illustrated and described prior to this re

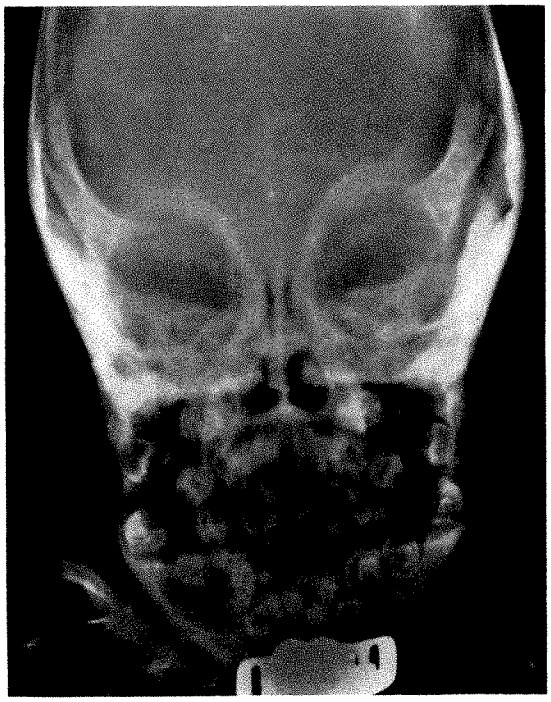


FIG. 4. U1442036 Eight year old while male with typical and severe changes of gargoylism as well as a positive urinary chondroitin test. The frontal roentgenogram of the skull demonstrates the "square," broad appearance of the mandible. Note also the abnormal dentition and poor sinus development.

port. Jervis,[9] in describing the skeletal changes associated with gargoylism, stated that there were "poorly formed articulating surfaces observed at best in the mandibular fossa and the acetabula." However, the appearance of the condyles was not illustrated or described in further detail and no reference was made to the specificity of the change. Lindsay and coworkers'[11] Case 2 is stated to have had rudimentary condyles, but no illustration or additional description of the condylar abnormality was presented. Pendergrass *et al.*[13] state that the ascending mandibular rami are underdeveloped, but no specific alteration of the mandibular condyles is mentioned. In Harvey's[4] report Figure 2 of the skull demonstrates the condylar change, but no comment regarding this abnormality is made in his otherwise excellent description of the skull changes associated with gargoylism.

The roentgenographic appearance of the mandibular condyles in older patients is unknown since longitudinal studies of these patients were not available. However, this abnormality probably persists into adult life in those patients who survive, as evidenced by illustrations in several other publications.[1,2]

On many occasions there is overlap roentgenologically between the various skeletal dystrophies and this makes their differentiation difficult. This is especially true with respect to gargoylism and Morquio's disease. In milder cases the skeletal changes may be similar and impossible to differentiate. However, the condylar configuration occurring in gargoylism in our experience has been a specific finding and, when present, permits a definite diagnosis.

Smith *et al.*[15] state that gargoylism "presents diagnostic roentgenographic features, particularly in the skull." This opinion was based primarily on the configuration of the sella turcica which has been shown to be a nonspecific although constant finding in

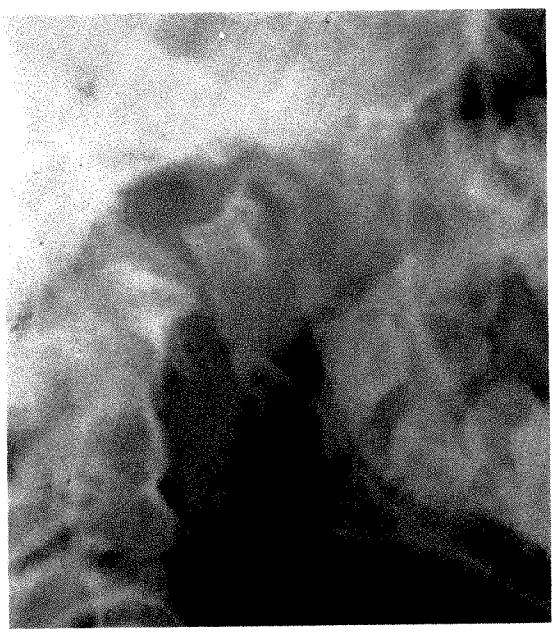


FIG. 5. U1441480 Nine year old white male with characteristic facies and skeletal features of gargoylism. The lateral roentgenogram shows the concave articular surface of the mandibular condyle.

gargoylism. On the other hand, we would agree with their statement, but on different grounds. The patient with classic clinical and roentgenographic features of gargoylism will present diagnostic changes in the skull also. These changes consist of dolichocephaly, prominent interdigitations along the coronal suture, elongation of the sella turcica with a long anterior recess beneath the anterior clinoid processes, prominent supraorbital ridges with depression of the bridge of the nose, retardation of sinus development with failure of development of the frontal sinus, square configuration of the mandible and a flattened or concave superior surface of the mandibular condyle. All of these findings, however, are nonspecific with the exception of the last. It should be stated that this abnormality, like the other skeletal features, develops after the first year of life and has not been seen in any patient younger than one year.

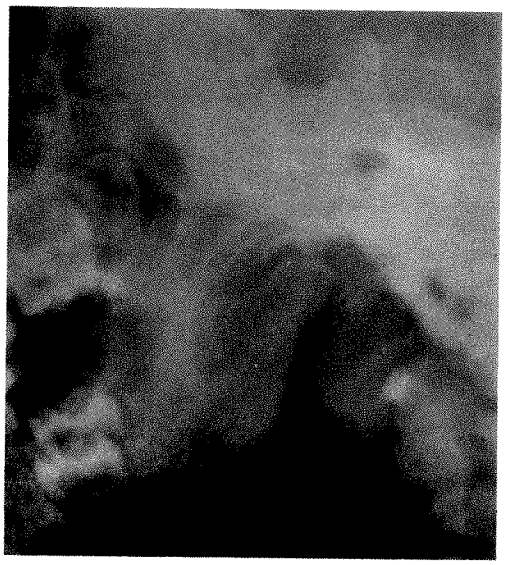


FIG. 6. U296608 Two year old white female with mild clinical and roentgenographic manifestations of gargoylism. The lateral roentgenogram of the skull demonstrates slight flattening of the mandibular condyle.

SUMMARY

1. The skull roentgenograms of 20 patients with gargoylism have been reviewed and a new finding has been described.

2. Concavity or flattening of the articular surface of the mandibular condyle has been observed to be a specific finding.

W. D. Horrigan, M.D.
St. Peter's General Hospital
New Brunswick, New Jersey

REFERENCES

1. CAFFEY, J. Gargoylism (Hunter-Hurler disease, dysostosis multiplex, lipochondrodystrophy); prenatal and neonatal bone lesions and their early postnatal evolution. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1952, *67*, 715–731.
2. CAFFEY, J. Pediatric X-ray Diagnosis. Third edition. Year Book Publishers, Inc., Chicago, 1956.
3. GILBERT, E. F., and GUIN, G. H. Gargoylism. *Am. J. Dis. Child.*, 1958, *95*, 69–80.
4. HARVEY, R. M. Hurler-Pfaundler syndrome (gargoylism). AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *48*, 732–740.
5. HENDERSON, J. L. Gargoylism. *Arch. Dis. Child.*, 1940, *15*, 201–214.
6. HUBENY, M. J., and DELANO, P. J. Dysostosis multiplex. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *46*, 336–342.
7. HUNTER, C. Rare disease in two brothers. *Proc. Roy. Soc. Med.*, 1916–1917, *10*, 104–116.
8. HURLER, G. Ueber einen Typ multipler Abartungen vorwiegend am Skelettsystem. *Ztschr. f. Kinderh.*, 1920, *24*, 220–234.
9. JERVIS, G. A. Gargoylism (lipochondrodystrophy). *Arch. Neurol. & Psychiat.*, 1950, *63*, 681–712.
10. LARSON, S., and LICHTY, J. A., JR. Gargoylism, report of three probable cases. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *50*, 61–66.
11. LINDSAY, S., REILLY, W. A., GOTHAM, T. J., and SKAHEN, R. Gargoylism; study of pathologic lesions and clinical review of 12 cases. *Am. J. Dis. Child.*, 1948, *76*, 239–306.
12. MEYER, K., GRUMBACH, M. M., LINKER, A., and HOFFMAN, P. Excretion of sulfated mucopolysaccharides in gargoylism. *Proc. Soc. Exper. Biol. & Med.*, 1958, *97*, 275–279.
13. PENDERGRASS, E. P., SCHAEFFER, J. P., and HODES, P. J. The Head and Neck in Roentgen Diagnosis. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1956.
14. REILLY, W. A., and LINDSAY, S. Gargoylism (lipochondrodystrophy). *Am. J. Dis. Child.*, 1948, *75*, 595–607.
15. SMITH, E. B., HEMPELMANN, T. C., MOORE, S., and BARR, D. P. Gargoylism (dysostosis multiplex): two adult cases with one autopsy. *Ann. Int. Med.*, 1952, *36*, 652–667.

ILIAC HORNS: A PATHOGNOMONIC ROENTGEN SIGN OF FAMILIAL ONYCHO-OSTEODYSPLASIA*

By CHARLES ZIMMERMAN, CAPTAIN, USAR (MC)
AUGUSTA, GEORGIA

HORN-LIKE osseous protrusions from the iliac bones are seldom encountered in daily radiologic practice. Even when these "iliac horns" are noted, their specific diagnostic significance may not be appreciated. It is the author's purpose to present examples of this entity and to pathognomonically relate it to an unusual familial onycho-osteodysplasia.

ILLIAC HORNS

Iliac horns are bilateral, not necessarily symmetric, osseous prominences which protrude posteriorly and laterally from the dorsal aspects of human iliac bones (Fig. 1 and 2). They are not known to occur in other mammals or lower forms.[5] These horns may be clinically palpable and their variable size is not related to the age or sex of the patients in whom they have been described. In many of the younger individuals, epiphyses may be seen capping the structures (Fig. 5 and 6). Opacification of pelvic muscle planes with injected contrast medium[2] suggests that these horns are not located at the origin or insertion of regional musculature, as is the case with osteocartilaginous exostoses.

It is striking that the reported patients with iliac horns have had associated characteristic bone or nail abnormalities or both. Further, the reported individuals belonged to families containing members with similar changes. Two recently observed illustrative cases are described.

REPORT OF CASES

CASE 1. A forty-one year old white female presented with a history of lower abdominal pain of two days' duration. This subsequently disappeared and the patient has been well since.

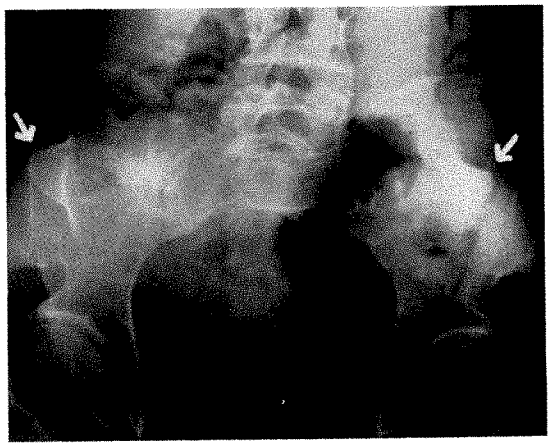


FIG. 1. Case 1. Note the slightly asymmetric "iliac horns." Also present are iliac bone hypoplasia, coxa valga and supra-acetabular notching

There was no history of previous major illness.

Because of pelvic findings on the abdominal roentgenogram the patient was recalled and the following physical findings, present since birth, were noted: (a) fingernail dystrophy (Fig. 3), most marked in the thumbnails but involving the other fingers and sparing the toenails; (b) bony prominences of both elbows with partial limitation of extension and flexion; and (c) palpable prominences in the region of the buttocks.

Roentgen Findings. Roentgenograms of the abdomen and pelvis revealed bilateral iliac bone protrusions which projected posteriorly in the oblique views. Unusual flattening of the outer aspects of the iliac bones was noted as well as supra-acetabular notching and coxa valga hip deformities (Fig. 1 and 2). Roentgenograms of the elbows (Fig. 4) revealed marked bilateral humeral capitulum underdevelopment and radial head luxation. Some hyperplasia of the olecranon process was present. Roentgen studies of the chest, knees and ankles were normal. Roentgenography of the hands revealed a stubby appearance of the ulna and radial styloids.

* This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication if this review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.

Comment. The patient's two younger daughters were subsequently examined and no abnormalities were found. (Her oldest daughter is described below.) There are no known similar abnormalities in the patient's parents, grandparents and siblings. Her husband was not available for examination but has no known nail or bone abnormalities.

Case II. This patient is a healthy thirteen year old white female, the oldest daughter of Case I, who was examined after her mother's findings were noted. Present since birth were the following physical findings: (a) fingernail dystrophy (Fig. 7), most severely affecting the thumbs with lesser involvement of the index fingers and sparing the toenails; and (b) partial limitation of elbow extension and flexion.

Roentgen Findings. Roentgenograms of the pelvis revealed bilateral small iliac horns capped by ununited epiphyses (Fig. 5 and 6). Supra-acetabular notching, flattening of the iliac bones and bilateral coxa valga hip deformities were present, changes similar to those of the mother. Roentgen studies of the elbows revealed humeral capitulum hypoplasia and radial head hyperplasia (Fig. 8). Roentgenograms of the chest, hands, knees and ankles were normal.

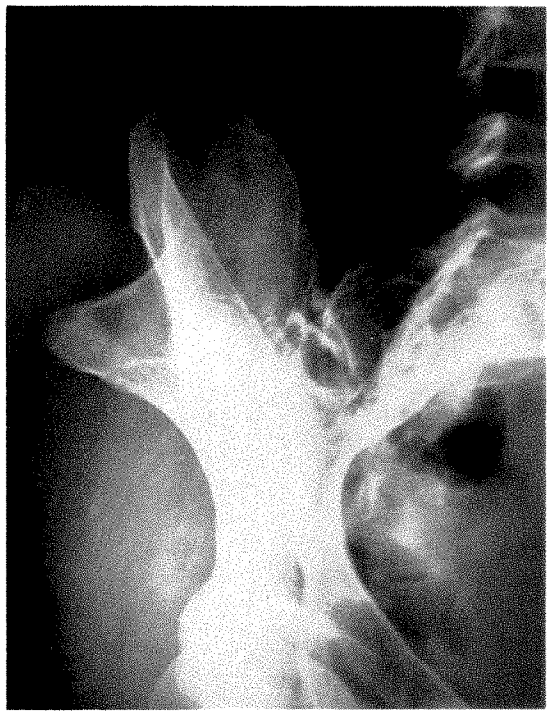


Fig. 2. Case I. An oblique view reveals the right-sided bony prominence projecting posteriolaterally. Similar findings are present on the left.

DISCUSSION

In our material, as with previously reported cases, iliac horns were discovered either as an accidental finding in a patient with unrelated complaints or at the subsequent examination of asymptomatic relatives.

CLASSIFICATION OF THE CHANGES

Three general groups of changes have been associated with iliac horns: (A) nail

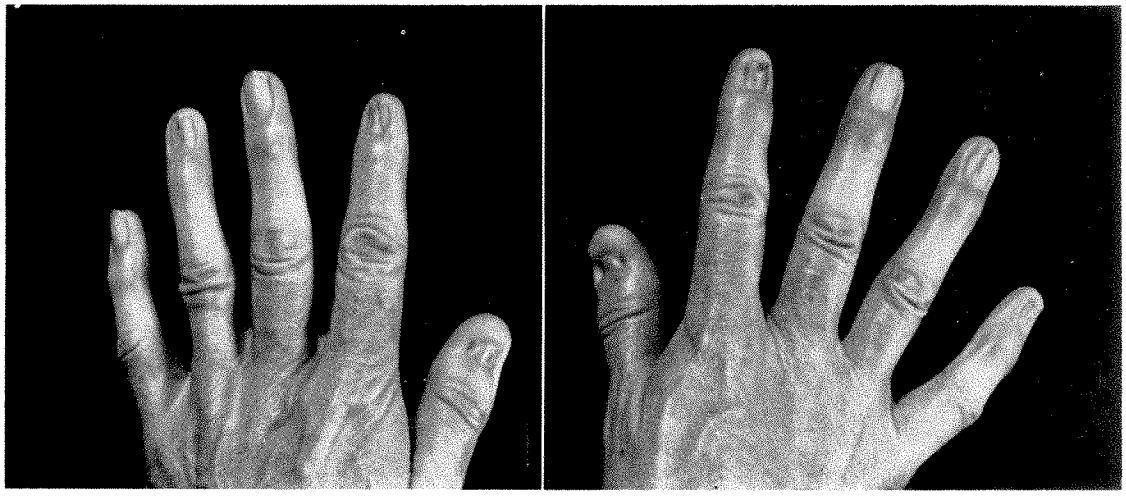


Fig. 3. Case I. Marked thumbnail dystrophy with lesser involvement of the index fingers, ring fingers and little fingers.



Fig. 4. Case 1. Right elbow. Hypoplasia of the capitulum humeri and hyperplasia of the medial epicondyle, with luxation and hyperplasia of the radial head.

dystrophies, (B) bone dysplasias, and (C) miscellaneous abnormalities.

A. *Nail Dystrophies.* The thumbnails often present with small normal bases which thin and tend to disappear distally,

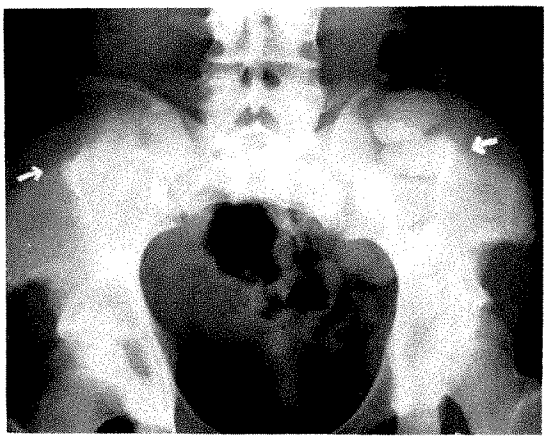


Fig. 5. Case II. Note the small bilateral "iliac horns," partially obscured by bowel on the left. Also present are iliac bone hypoplasia, coxa valga and supra-acetabular notching.



Fig. 6. Case II. Close-up of the right ilium revealing a small iliac horn capped by epiphysis. There was a similar finding on the left side.

merging with the fleshy tips of the fingers so that there are no free ends of the nails. Other fingernails and the toenails are less often involved and may be marred by longitudinal ridges and lateral concavities (Fig. 3 and 7).

B. *Bone Dysplasias.* The involved osseous structures present with various hypoplasias or hyperplasias. Elbow and knee joint bones are most commonly involved. The capitulum humeri is hypoplastic (Fig. 4 and 8) and there is a tendency to radial head luxation, hypoplasia or hyperplasia. Hyperplasia of the coronoid process of the ulna and of the medial epicondyle of the humerus may occur. Variable degrees of limitation of elbow flexion and extension are common. Agenesis or hypoplasia of the patella is often present with a tendency to habitual dislocation. There may be hyperplasia of the femoral condyle and continuous hypoplasia of the tibial plateau. Although knee abnormalities are commonly seen, they were not present in our cases.

In addition to iliac horns, the pelvis may present with any or all of the following: hypoplastic iliac wings, supra-acetabular notching, protrusio acetabuli and coxa valga. Other minor periarticular hypoplasias or hyperplasias have been reported and are summarized elsewhere.[8]

C. *Miscellaneous Abnormalities.* An irregular zone of pigmentation separating the

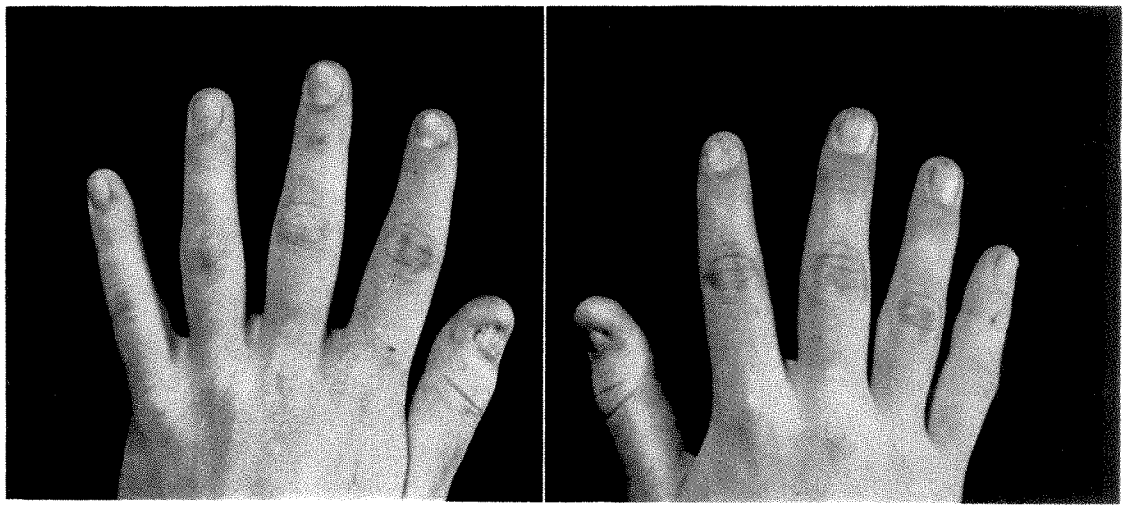


Fig. 7. Case II. Marked thumbnail dystrophy with lesser involvement of the index fingers.

pupil from the iris is uncommonly associated with iliac horns.[6,10] "Hereditary glomerulonephritis" has been reported in a family with iliac horns and the above changes.[5] In 1 reported case, nonunion of a simple forearm fracture was considered a manifestation of generalized bone abnormality.[8]

All or some of the above general groups of changes have been found in patients with iliac horns and in their families. Generally, no significant incapacity is associated, although some did have osteoarthritides, which may have been secondary to the periarticular dysplasias. It should be noted that the nail and bone changes commonly occur in relatives of the reported cases, even though iliac horns are absent.

REPORTS IN THE LITERATURE

As a matter of interest, an unusual familial entity characterized by all or some of the knee and elbow dysplasias and nail dystrophies has been known since the last century[7] and has been repeatedly described.[8] Of radiologic significance is the fact that Turner,[14] in reporting two families with these changes, included a pelvic roentgenogram clearly showing iliac horns. He, however, made no mention of this finding. Lester,[6] reported another family and mentions, without illustrative studies, that the "crest of the ilium seems enlarged posteriorly." Fong[4] briefly described a patient with iliac horns but apparently little clinical information was available to him.

Mino, Mino, and Livingstone[9] were the

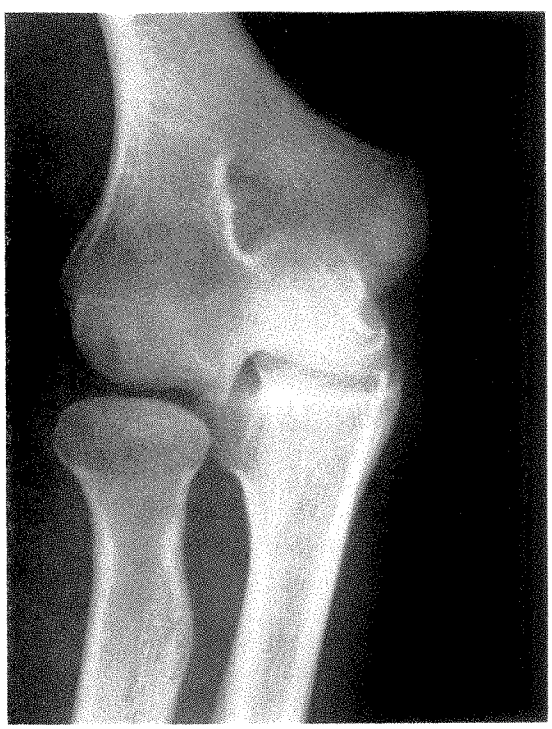


Fig. 8. Case II. Right elbow. Hypoplasia of the capitulum humeri and hyperplasia of the radial head.

first to relate iliac horns to the previously known onycho-osteodysplasia. Since then, iliac horns have been described by Thompson, Walker and Weens,[12] Hawkins and Smith,[5] Böck,[2] Roeckerath,[10] Cottet and Pfändler,[3] Wedler and Welsch,[15] Bates,[1] Toison, Guillemet and Carlier,[13] and by Mangini[8] in 36 individuals, whose ages have ranged from nine months to sixty-five years. In no case has the observation of iliac horns been an isolated finding and either one or more manifestations of onycho-osteodysplasia have been associated.

GENETICS

The hereditary mechanism involved in these unusual ectodermal and mesodermal abnormalities is far from clear.

One hypothesis[11] implicates a single polyphenotypic dominant nonsex-linked gene which is able to cause many deformities in varying degrees. Such a mechanism is the generally accepted one in other abnormalities, *e.g.*, osteogenesis imperfecta. Another theory[10] suggests that partial or complete manifestations of the complex are best explained by many genes on one chromosome with the resultant effect dependent upon their closeness. Some even deny a hereditary mechanism and suggest alterations in the maternal biochemical or physiologic milieu. Supporting this opinion is the ability of investigators to consistently obtain skeletal and soft tissue anomalies in the offspring of female rats maintained on riboflavin deficient diets.[9] Still another point of view[8] maintains that two pathologic genes are present, one determining nail dystrophy and the other influencing arthrodysplasia, but that their net effect is influenced by interactions with several environmental factors, such as metabolic errors and accidents. The result is that some family members do not have stigmata of the disease while others have varying manifestations.

Chondroectodermal dysplasia (Ellis—Van Crevald) can also involve nails and bony structures such as the pelvis and elbows. However, the absence of knee changes or familial incidence and the marked dentition abnormality indicate an unrelated entity.

SUMMARY

Iliac horns are a pathognomonic roentgen sign of familial onycho-osteodysplasia. Their discovery should alert the radiologist to initiate a search for characteristic unguinal and osseous changes in the patient and his family.

Two related cases with iliac horns and onycho-osteodysplasia are presented.

The literature is reviewed and possible genetic mechanisms are discussed.

129 Woodland Avenue
Maywood, New Jersey

The author wishes to express appreciation to Dr. H. J. Peters, Maj. V. L. Oddi and Sgt. W. Miller for assistance with translations and to Mr. G. E. Peters, R.T., for roentgenograms and photographs.

REFERENCES

1. BATES, J. C. Iliac horns; manifestation of hereditary osteo-onychodysplasia. *U. S. Armed Forces M. J.*, 1954, *5*, 865–871.
2. BÖCK, K. Beckenhorner, eine angeborene erbliche Anomalie im Rahmen eines Missbildungssyndroms. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1951, *74*, 543–549.
3. COTTET, P., and PFÄNDLER, U. Hereditary osseous and ungual dysplasia. *Acta radiol.*, 1952, *37*, 111–120.
4. FONG, E. E. "Iliac horns" (symmetrical bilateral central posterior iliac processes); case report. *Radiology*, 1946, *47*, 517–518.
5. HAWKINS, C. F., and SMITH, O. E. Renal dysplasia in family with multiple hereditary abnormalities including iliac horns. *Lancet*, 1950, *1*, 803–808.
6. LESTER, A. M. Familial dyschondroplasia associated with anonychia and other deformities. *Lancet*, 1936, *2*, 1519–1521.
7. LITTLE, E. M. Congenital absence or delayed development of patella. *Lancet*, 1897, *2*, 781–784.
8. MANGINI, U. Una rara sindrome ereditaria e familiare "corni iliaci" associati a displasie articolari e ungueali e ad altre varie deformita. *Arch. "Putti" chir. org. movimento*, 1955, *6*, 325–344.
9. MINO, R. A., MINO, V. H., and LIVINGSTONE, R. G. Osseous dysplasia and dystrophy of nails; review of literature and report of case.

Am. J. Roentgenol. & Rad. Therapy, 1948, *60*, 633–641.

10. Roeckerath, W. Hereditäre Osteo-onycho-dysplasie. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1951, *75*, 700–712.
11. Senturia, H. R., and Senturia, D. B. Congenital absence of patellae associated with arthrodysplasia of elbow and dystrophy of nails; hereditary syndrome. Am. J. Roentgenol. & Rad. Therapy, 1944, *51*, 352–358.
12. Thompson, E. A., Walker, E. T., and Weens, H. S. Iliac horns; osseous manifestation of hereditary arthrodysplasia associated with dystrophy of fingernails. *Radiology*, 1949, *53*, 88–92.
13. Toison, J., Guillemet, M., and Carlier, C. Malformations osseuses multiples avec cornes iliaques. *J. de radiol. et d'électrol.*, 1955, *36*, 386–388.
14. Turner, J. W. Hereditary arthrodysplasia associated with hereditary dystrophy of nails. *J.A.M.A.*, 1933, *100*, 882–884.
15. Wedler, H. W., and Welsch, A. Zur Morphologie des Turnerschen Syndroms. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1952, *76*, 586–591.

ROENTGENOLOGIC ASPECTS OF HIP LIMP IN CHILDHOOD*

By R. W. CARTER, M.D.
BIRMINGHAM, ALABAMA

THE problem of hip limp in childhood is one which is encountered by the general practitioner, pediatrician and orthopedist as well as the radiologist. The causes of limp are numerous, ranging from such diverse conditions as improperly fitting shoes and behavior problems to disorders that produce permanent crippling if not treated early and adequately.

The purpose of this paper is not to present an exhaustive list of everything which might produce limp, or to demonstrate roentgen findings in gross, far advanced cases, but rather to emphasize the early and minimal findings. Limp due to neurogenic causes is not included in the discussion.

The abnormalities found in the growing hip constitute an interesting group of conditions which commonly present themselves as limp, either painful or painless, or as pain referred to the knee or anterior thigh. Early diagnosis is important in all of these ailments, and in some of them it is critical.

Roentgenograms of high quality, showing both bone and soft tissue detail, are essential. Both hips (in anteroposterior and lateral projection) should be routinely examined. This is because of the frequency of bilateral involvement and the desirability of comparing sides as an aid in detecting minimal changes. Taking an anteroposterior roentgenogram of the pelvis, to include both hips, and a lateral roentgenogram of the hips in "frog leg" position is a good routine practice.

These conditions are presented in the order in which they are usually seen, starting with the defects found in younger patients and progressing to those in older patients.

CONGENITAL DISLOCATION OF THE HIP

Ideally, congenital dislocation of the hip should not be included in the causes of hip limp in childhood. It should be diagnosed by physical examination shortly after birth; a diagnosis made after the child has started to walk is a late diagnosis.[6] Bilateral congenital dislocation of the hip is more difficult to diagnose clinically than the unilateral cases because the abnormal findings are symmetric and more easily overlooked.

On examination, the skin folds are seen to be asymmetric, the infant is reluctant to use the affected extremity and there is shortening of the involved extremity. When the patient lies flat on his back with both hips and knees flexed, the affected leg appears shorter and will resist full abduction. After the child begins to walk, there is a painless limp in unilateral cases. In bilateral cases the gait is said to be typical and is waddling in type. There is a prominent abdomen and increased lumbar lordosis since the line of weight-bearing is shifted posterior to the acetabulum.

Congenital dislocation of the hip is approximately six times more common in female than in male infants. It is more often unilateral, and is very rare in Negro infants. In Asiatic countries, where the child is carried on the mother's hip, with the legs abducted, the incidence of congenital dislocation is reduced.[3]

On roentgen examination, before the appearance of the capital femoral epiphysis, the increased slant of the acetabular roof may be the only abnormal finding; displacement of the femoral shaft upward and laterally may also be noted. Interpretation of the former finding is difficult, since increased slant of the acetabular roof does

* From the Department of Radiology, Norwood Clinic and Carraway Methodist Hospital, Birmingham, Alabama.

not always mean that the child has or will develop congenital dislocation of the hip.[1]

After the appearance of the capital femoral epiphysis, usually at about the age of six months, recognition is easier. In congenital dislocation there may be delay in the appearance of the capital femoral epiphysis and, once it appears, it may be smaller than normal. Comparison with the opposite side is helpful.

The radiologist should consider the possibility of congenital dislocation in cases of increased slope of the acetabular roof, lateral and upward displacement of the femoral shaft, or asymmetric or underdevelopment of the capital femoral epiphysis. These suggestive roentgen findings, when correlated with the physical findings, offer the optimum chance for early diagnosis (Fig. 1).

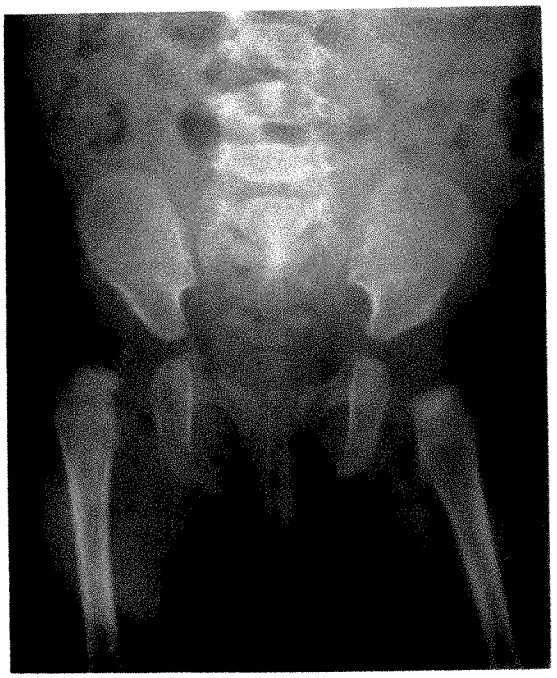


Fig. 1. Congenital dislocation of the right hip. Neither capital femoral epiphysis has appeared. There is increased slope of the right acetabulum and displacement of the femoral shaft laterally and cephalad.

COXA VARA

This term refers to a condition in which there is a decrease in angulation between the femoral neck and shaft. Normally, the angle of the femoral neck is 120–130 degrees, but in coxa vara it may be 60 degrees or less. The defect may be congenital or acquired. Very little is known of the pathology of the congenital type. It is assumed to be due to anomalous ossification of the proximal third of the femur. Acquired types may be associated with rickets, osteomalacia and fractures of the femoral neck (either traumatic or pathologic) and other causes.

The limp associated with coxa vara is painless. It may appear in children learning to walk, or it may have an insidious onset in later years following minor trauma.

The physical examination shows limitation of abduction and internal rotation. In bilateral cases, as with congenital dislocation, there is increased lumbar lordosis and a protuberant abdomen. The femoral head is palpable, however, in its normal location and there is no telescoping. Clinically, coxa vara is similar to congenital dislocation except for these latter two findings.

This condition is usually easy to diagnose on roentgen examination, which shows the decreased angulation between the femoral neck and shaft as described. If the condition is acquired, or associated with another disease, other stigmata may be seen on the roentgenograms of the pelvis or other parts (Fig. 2).

PYOGENIC INFECTION OF THE HIP

This condition may appear a few days following birth and most cases are observed in infants. It does not commonly produce limp, since the child is usually too ill or too young to walk. If the hip joint is to be saved, prompt diagnosis is essential. There is early destruction of the joint cartilage by proteolytic enzymes produced by the pyogenic organisms. It may start as an osteomyelitis of the femoral neck and, because the femoral neck is contained within the joint capsule, spread to the joint itself.

The patients usually appear acutely ill, but not invariably so. There is marked

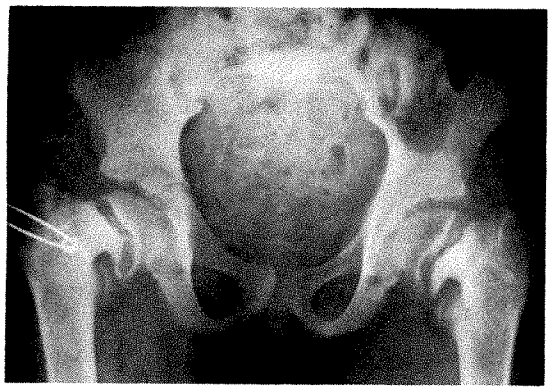


FIG. 2. Congenital bilateral coxa vara. There is a marked decrease in the angle between the femoral shaft and neck. This is a typical finding and usually makes possible an easy roentgen diagnosis.

limitation of motion, fullness about the hip joint, fever and leukocytosis. This condition usually presents an acute fulminating picture as opposed to tuberculosis of the hip, which is usually insidious and not common in such young infants. The hip infection may be secondary to infection elsewhere, as in the urinary tract, respiratory tract or skin. However, it may also be primary within the hip itself.

On roentgen examination there is widening of the joint space and prominence of the shadow of the joint capsule. Areas of destruction in the femoral neck may appear later. Destruction may be greater in degree than it appears on the roentgenogram because so much of the femoral head and neck is cartilage in infants.[3] In advanced cases actual dislocation of the joint may occur.

TUBERCULOSIS OF THE HIP

The hip is the most common joint involved by tuberculosis and constitutes 20 per cent of cases of bone and joint tuberculosis.[3]

The tubercle bacillus reaches the joint by hematogenous spread and may primarily involve the synovia or inferior neck adjacent to the epiphyseal line, but it is best to consider it as a general involvement of the joint.

The condition has an insidious onset with hip pain, limp, atrophy of leg muscles, afternoon fever and, frequently, night cries. As in most causes of childhood limp, the pain may be referred to the knee or thigh with little or no complaint of hip pain. Pain is often worse on arising but improving with activity, only to recur following longer periods of exercise. If untreated, after weeks or months activity becomes impossible and the patient is bedridden.

The tuberculin test is practically always positive and a negative tuberculin test with a suspected tuberculous hip should lead to checking of the antigen and use of another source.[3] Tuberculous infection of the hip is infrequent under two years of age.

On early roentgen examination, there is widening of the joint space due to increased joint fluid and decreased density of the bones of the hip, extending into the proximal portions of the neck of the femur. Later, the joint space is narrowed. Although there are no proteolytic enzymes associated with tuberculous infection, progression of the disease is so slow that the narrowing of the joint space stands out. There is prominence of the capsular shadow; trabeculae become less prominent and have a hazy appearance. The disease may progress to gross bone destruction (Fig. 3).

SYNOVITIS OF THE HIP

This is a very important and probably the most common cause of hip limp in childhood. It bears a poorly understood relationship to other more serious hip diseases in children and adults. Children with synovitis of the hip have an increased probability of developing Legg-Perthes' disease and synovitis appears at times to be the first symptom of an incipient form of the disease. It may be an underlying cause in any unilateral degenerative hip disease discovered in the second decade of life.

The etiology is unknown. Trauma, allergy, and toxic factors have been postulated, but none of these factors can apply to all cases.[2]

Clinically, the condition closely mimics Legg-Perthes' disease. The limp, age group and physical findings are very similar.

It has been called the most common cause of irritable hip in the older child with a negative roentgen examination.[5] A close study of good quality roentgenograms, however, will reveal positive findings. The soft tissue shadow of the joint capsule is thickened and bulges laterally and superiorly. Comparison with the opposite side is helpful. Soft tissue swelling of muscles (obturator internus, gluteus minimus and iliopsoas) is a less constant finding and more difficult to interpret. There may also be slight widening of the joint space due to an increase in fluid. This is more easily detected medially[2] (Fig. 4).

The symptoms usually subside rapidly with treatment and this also aids in differentiating synovitis from other clinically similar hip conditions.

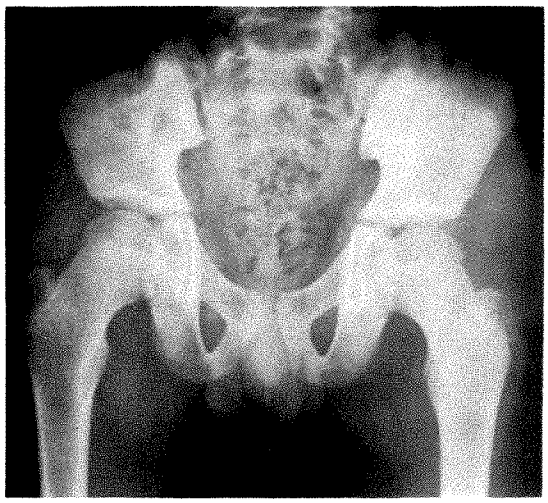


Fig. 4. Synovitis of the right hip. There is marked lateral bulging of the capsule of the right hip. The capsule of the left hip joint appears normal.

Legg-Perthes' disease

This is a common and serious cause of hip limp in children. It is the most common of the osteochondroses. It occurs primarily in a four to twelve year age group.

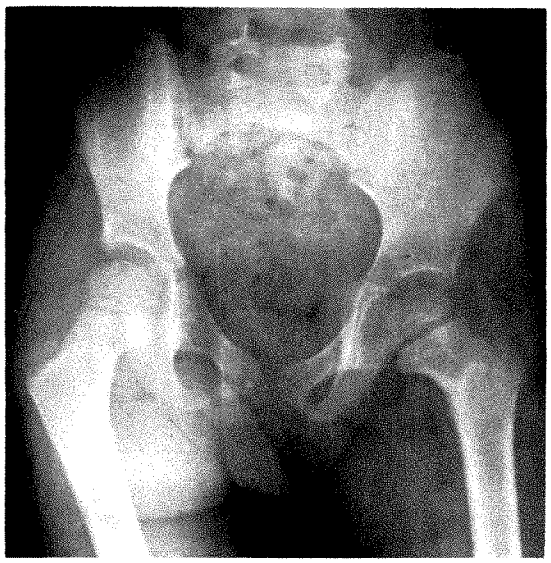


Fig. 3. Tuberculosis of the left hip. There is widening of the joint space medially and widening of the epiphyseal line. An ill-defined area of destruction is noted in the femoral neck. At surgery a reddish-brown fluid spurted out when the capsule was opened. Culture revealed tubercle bacillus.

Like in other types of osteochondrosis, the basis for this disease appears to be circulatory impairment of the growing bone. There is involvement of the capital femoral epiphysis with sequestration of this area followed by gradual regrowth over a period of years. The condition is approximately 8 to 1 times more common in males, suggesting that physical activity may have some relation to the cause.

It is important to examine both hips because of the frequency of bilateral involvement. If, initially, the disease is unilateral, the opposite hip should be observed at intervals during treatment for possible beginning involvement. As mentioned above, there is sometimes preceding or associated synovitis. Cases observed at surgery have shown evidence of synovitis.

Clinically there is a history of limp, with pain at the knee or hip, for a period of three weeks to two months. There may be atrophy of the thigh muscles on the involved side. Symptoms are not always present from the beginning of the disease. Some cases are seen with a short duration of symptoms and advanced roentgen changes. The insidious onset sometimes simulates

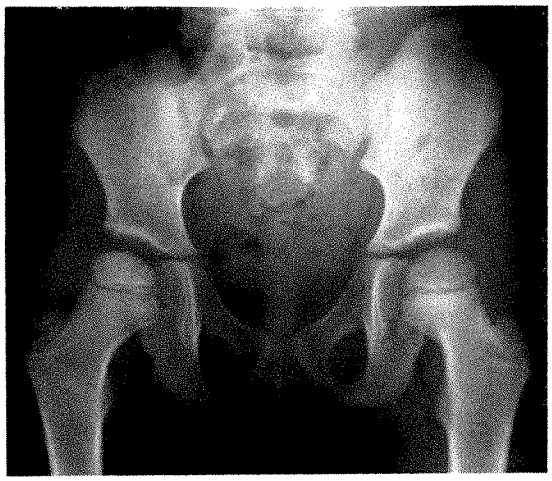


FIG. 5. Legg-Perthes' disease of the right hip. There is slight rarefaction of the right femoral neck and minimal widening of the epiphyseal line with some irregularity and flattening of the right capital femoral epiphysis.

tuberculosis of the hip as well as other chronic hip lesions.

The earliest roentgen sign is widening of the epiphyseal line and irregularity of the metaphysis of the femoral neck. The articular capsule may be prominent. Rarefaction of the femoral neck occurs. Later there is flattening of the femoral head with the neck becoming short and thick, and the capital femoral epiphysis has a sclerotic, fragmented appearance (Fig. 5).

SLIPPED CAPITAL FEMORAL EPIPHYSIS

This condition occurs in a slightly older age group (nine to sixteen years); *i.e.*, preadolescent and adolescent children. It is commonly seen in obese and poorly developed children, although tall asthenic children may also develop slipping of this epiphysis.

The plane of the epiphyseal line changes from the horizontal to the oblique as the child becomes adolescent and it has been postulated that this makes the epiphysis vulnerable to a shearing force from the increased angulation.[1] This produces a chronic limp, which may or may not be painful. The pain is frequently referred to the knee. Clinically the limp shows little difference from the limp of Legg-Perthes' disease.

The early roentgen findings are similar to those in Legg-Perthes' disease, namely, widening of the epiphyseal line and rarefaction of the femoral neck. The slipping usually is downward and backward from the normal position, or it may also be medial. Klein *et al.*[4] have stated that a line drawn along the superior border of the femoral neck, on the anteroposterior roentgenogram, should transect the capital femoral epiphysis (Fig. 6). In cases of medial slipping this transection would not occur. Without attention to this detail, medial slipping could easily be overlooked. Posterior slipping is detected in the lateral view which usually demonstrates an obvious change in the relationship between the epiphysis and the femoral neck.

SUMMARY

1. Good quality roentgenograms and attention to minimal findings are necessary for early diagnosis of conditions causing hip limp in childhood.

2. Comparison with the opposite side is helpful in detecting early changes.

3. Early diagnosis is important in all

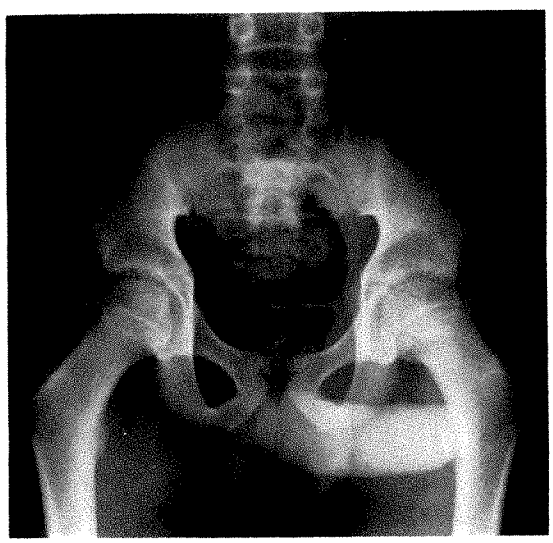


FIG. 6. Slipping of the left capital femoral epiphysis. Slight medial slipping on the left is noted; a line drawn along the superior border of the femoral neck would not transect the epiphysis, as it would on the right.

these conditions, and critical in some; any help the radiologist can give the clinician will increase the chance of earlier recognition.

Norwood Clinic
1529 North 25th Street
Birmingham, Alabama

REFERENCES

1. Caffey, J. Pediatric X-Ray Diagnosis. Third edition. Year Book Publishers, Inc., Chicago, 1956.
2. Donaldson, W. F. Transient synovitis of hip joint. *Pediat. Clin. North America*, 1955, *2*, 1073–1080.
3. Ferguson, A. B., Jr. Orthopedic Surgery in Infancy and Childhood. Williams & Wilkins Company, Baltimore, 1957.
4. Klein, A., Joplin, R. J., Reidy, J. A., and Hanelin, J. Roentgenographic features of slipped capital femoral epiphysis. Am. J. Roentgenol. & Rad. Therapy, 1951, *66*, 361–374.
5. Lloyd-Roberts, G. C. Limping child. *Brit. J. Clin. Pediat.*, 1959, *13*, 221–222.
6. Rapp, I. H. Limp in infancy and childhood. *North Carolina M. J.*, 1955, *16*, 57–61.

BILATERAL INCURVING OF THE TERMINAL PHALANGES OF THE FIFTH FINGERS*

AN ISOLATED LESION OF THE EPIPHYSEAL PLATE

By HERBERT J. KAUFMANN *and* WILLY F. TAILLARD
BASEL, SWITZERLAND

CONGENITAL anomalies involving the phalanges are, on the whole, easily recognized and classified. Particularly, the incurving of the fifth finger is well known as a stigma of mongolism or as an isolated anomaly occurring sometimes as a familial trait. There also exists, however, a condition of apparently acquired nature affecting the terminal phalanx of the fifth finger.

Kirner[3] is credited with the first description, in 1927, of this disorder. A few subsequent publications have been devoted to this entity, bringing the total number of cases reported so far to 14.[1,2,4–9] From the data available in these reports it appears justified to delineate this disorder in a clear fashion from all the other affections involving the phalanges of the fifth fingers. Since, in the American literature, there is no information available on this peculiar bony disturbance, we would like to report the findings of a case recently seen at the Children's Hospital of Basel, Switzerland, together with the data of a second case from the files of the X-ray Department of the Children's Hospital, Boston, Massachusetts.

CLINICAL FEATURES

From the accumulated data it can be seen that this disorder is first noticed in preadolescence, between the ages of eight and fourteen years. Girls appear to be affected more frequently than boys, in a ratio of about 2:1. According to the histories, trauma does not seem to play any role.

The onset is insidious with the parents usually reporting slow deviation of the terminal phalanx of the little fingers manifesting itself in a progressive manner over a period of many months or even years. There are no subjective complaints of pain or neurologic manifestations. The only difficulty a patient may encounter is in the execution of skilled acts requiring the use of the little fingers, such as, playing the violin or piano, or using a typewriter.

Objectively, there is usually minimal redness over the affected phalanx with some degree of swelling. However, no tenderness or hyperthermia can be noticed. In no instance were there signs of local infection. The position of the axis of the terminal phalanx of the fifth fingers is strikingly and uniformly abnormal. There is a considerable degree of palmar and radial deviation with an element of rotation (supination) of the most distal portion of the fifth fingers. The convexity of the fingernails may be abnormally pronounced.

ROENTGEN FEATURES

The deviation, as seen on the roentgenogram, does not affect the entire terminal phalanx but only its diaphysis. The epiphysis is in the normal position. The joint space between the middle and terminal phalanx is normally preserved. The metaphysis is ill defined, if visualized at all. In some cases, the epiphyses appear thicker than those of the unaffected terminal phalanges or fingers. The space of the epiphyseal plate is generally widened. The diaphysis deviates 10° to 50° radially from its normal axis and to almost the same degree in the palmar direction. The diaphyses are markedly narrowed; they

* From the Departments of Roentgenology and Orthopedic Surgery of the Children's Hospital of the University of Basel, Switzerland; and the Department of Radiology of the Children's Hospital Medical Center, Harvard University Medical School, Boston, Massachusetts.

Presented in part at the Annual Meeting of the Society for Pediatric Radiology in Atlantic City, September 26, 1960.

appear bent and show a loss of normal trabeculation. The cortical portions are often ill defined with an irregular contour, particularly on the palmar and radial sides. These changes can be present in varying degrees according to the stage of the disease process.

REPORT OF CASES

Case 1. This nine year old girl was seen at the Children's Hospital of the University of Basel. At the time of admission to the hospital for a diagnostic work-up for an unrelated disorder, it was noted that the terminal phalanges of the fifth fingers deviated symmetrically in a palmar and radial direction. She was the only child of healthy parents and there was no information available on any bony abnormalities in the father's or mother's families. The parents claimed that the abnormal shape and direction of the tips of both fifth fingers had been present for some time (several years?) but that the changes observed in early infancy were by no means as marked as at the present. At no time were there subjective complaints. Trauma of any kind was denied.



Fig. 1. Case 1. Dorsal view showing the incurved terminal phalanges of the fifth fingers. The swelling over the distal phalangeal joints is well visualized.

Physical Examination. Both terminal phalanges of the fifth fingers deviated radially by 40° to 50° (Fig. 1) and to a similar degree in the palmar direction when viewed in the lateral aspect. In the region of the distal joints of both the affected fingers, there was a mild degree of diffuse swelling and thickening of the skin together with minimal redness, but no local hyperthermia or pain to pressure. Skin sensitiv-

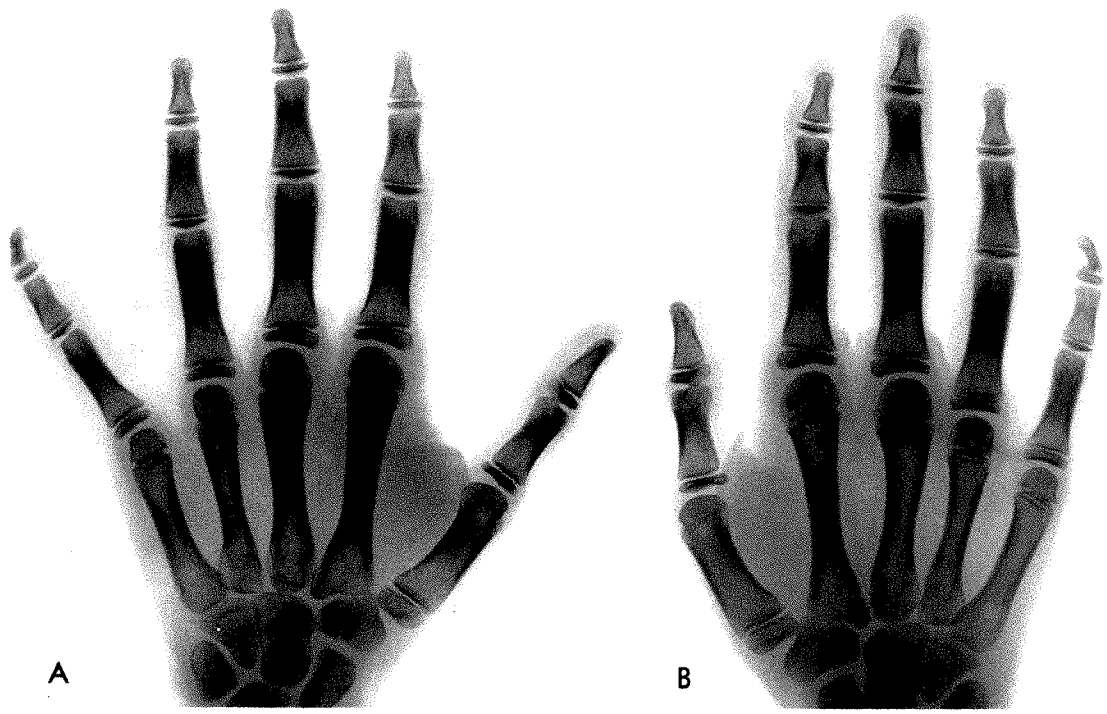


Fig. 2. Case 1. (*A* and *B*) Posteroanterior roentgenograms of both hands.

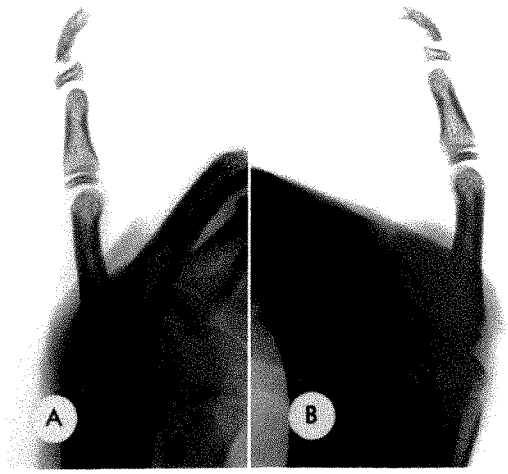


FIG. 3. Case I. (*A* and *B*) Lateral roentgenograms of the fifth finger of each hand.

ity and proprioception were normal. The range of motion of the joint was only mildly restricted in that neither complete extension nor complete flexion could be achieved.

Roentgen Findings. The only abnormal findings are limited symmetrically to both terminal phalanges of the fifth fingers (Fig. 2, *A* and *B;* and 3, *A* and *B*). The diaphysis is in an abnormal relationship to the epiphysis with widening of the epiphyseal plate. The trabeculation of the diaphysis is highly irregular with areas of increased density in the central portions and less definition of the cortex, particularly on the palmar and radial sides. Also there is marked structural loss of the metaphysis. No signs of osteolysis, bony sequestration or callus formation can be recognized. A skeletal survey revealed nothing abnormal.

In addition, roentgenograms of the feet of this patient and of the hands and feet of both her mother and father were obtained but they presented no abnormal findings.

CASE II. A ten year old boy was seen at the Children's Hospital, Boston, in the year 1944. He was brought to the hospital for a deformity of both fifth fingers. The mother claimed that during the preceding four years there had been gradual thickening and deviation of the terminal phalanges of the fifth fingers. At no time was there pain, local infection or any joint involvement. According to the history, there had been no significant disease. The boy was fully active with no subjective complaints.

Physical Examination. There was thickening of the terminal phalanges of the fifth fingers with minimal "clubbing" (Fig. 4, *A* and *B*); all other joints were normal. Temperature measurements by skin electrodes revealed no differences between the local affected areas and the rest of the fifth fingers or any portion of the other fingers or their terminal phalanges.

Roentgen Findings. No evident abnormality is present except for that of each fifth finger where there is considerable soft tissue thickening at the tip and considerable deformity of each distal phalanx (Fig. 5, *A*, *B* and *C*). These are irregular in contour, with considerably increased density and palmar and radial devia-

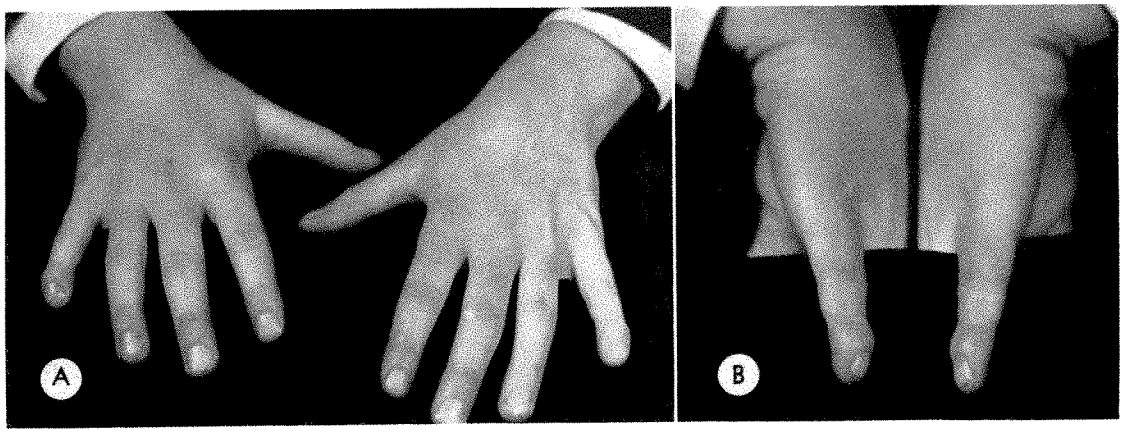


FIG. 4. Case II. (*A*) Photograph of the dorsal aspect of both hands demonstrates the shortness and incurving of the terminal phalanges of the fifth fingers. (*B*) Photograph in the lateral view of the fifth fingers shows the palmar deviation, diffuse swelling over the distal joints and the exaggerated convexity of the fingernails.

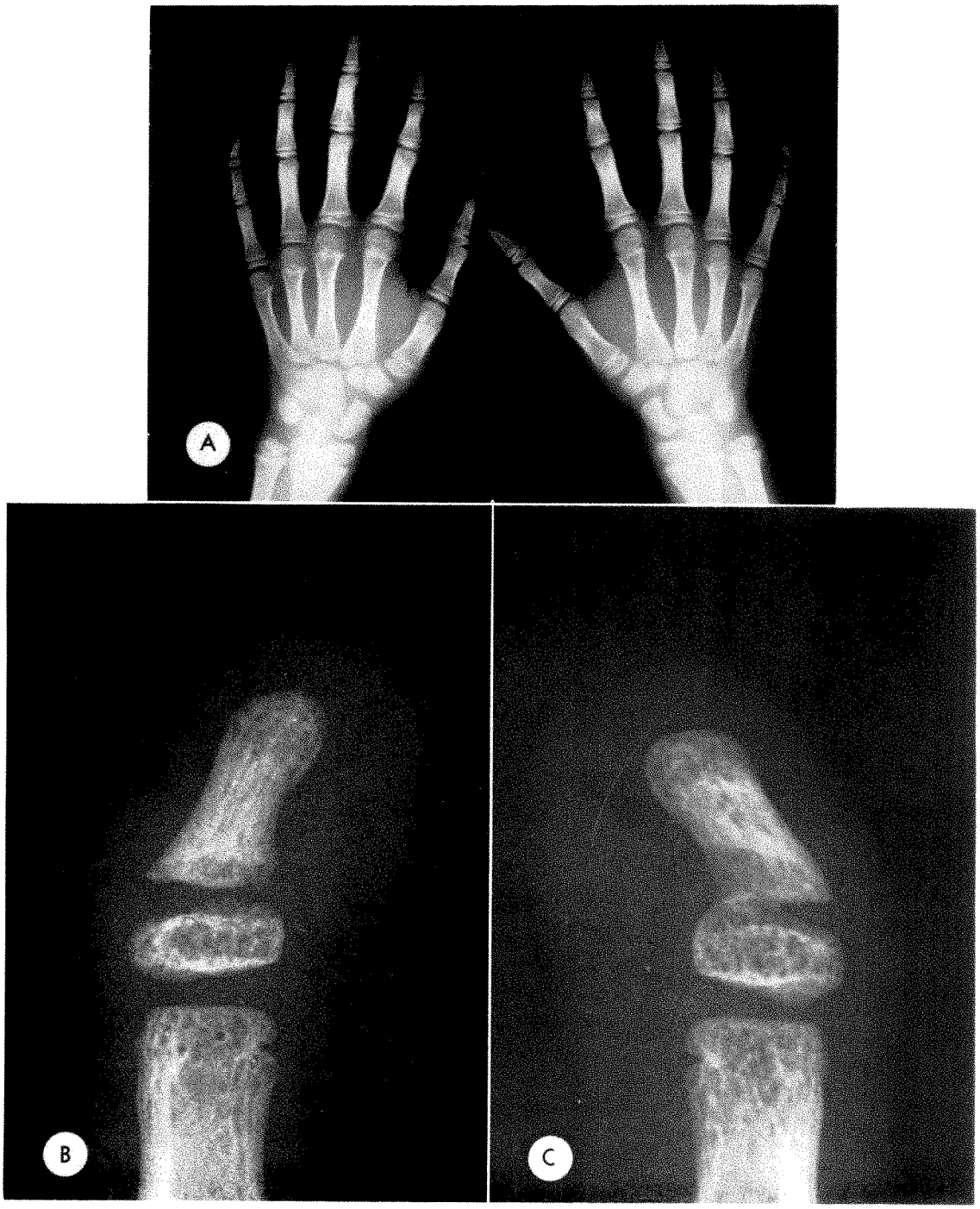


Fig. 5. Case II. (*A*) Posteroanterior roentgenogram of both hands. (*B* and *C*) Detailed views of the affected terminal phalanges.

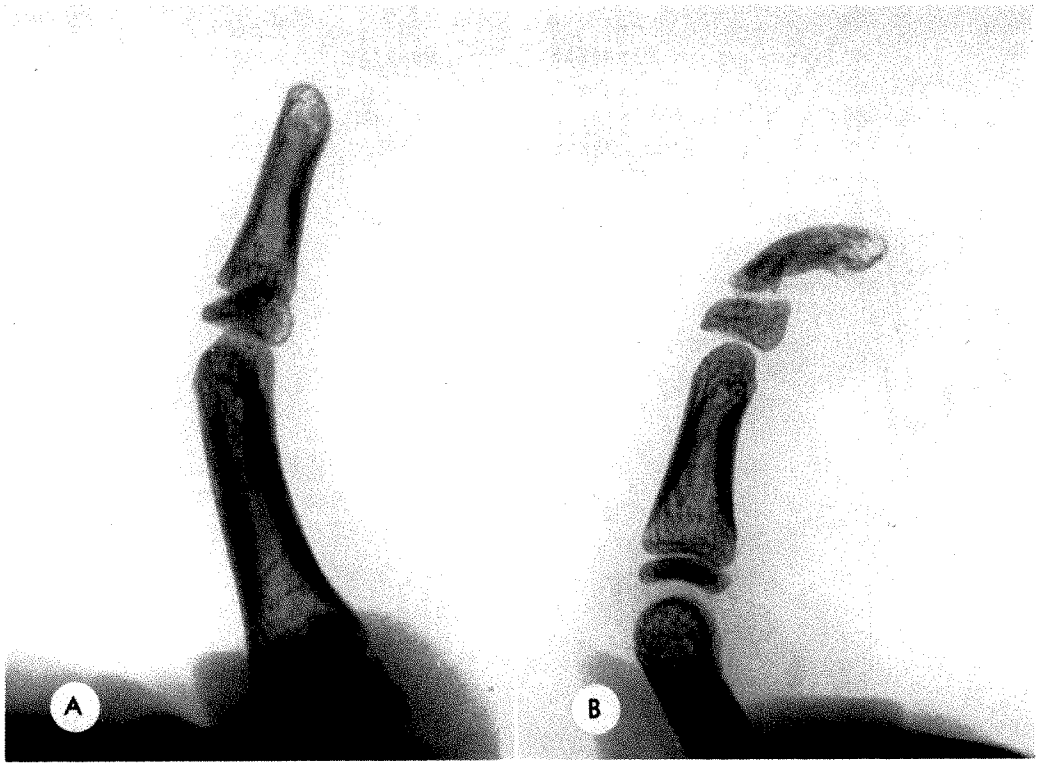


FIG. 6. Case 1. (*A* and *B*) Lateral views of the fourth and fifth fingers.

tion. There is nothing to suggest osteomyelitis. Examination of the long bones revealed no essential variation from the normal.

Comment. The etiology of these changes is not evident but they are such as those frequently associated with a vascular disturbance or conditions such as scleroderma. However, they also may be developmental in nature.

DISCUSSION

From these 2 cases and the 14 other cases recorded to date, the following characteristics of this disorder can be recognized: (1) manifestation between eight and fourteen years of age; (2) painless swelling and progressive palmar and radial deviation of the distal phalanges of the fifth fingers over a period of time (three months to several years); (3) bilateral involvement (in 14 of the 16 patients); (4) usually symmetric progression with, however, single cases showing a slight difference in the stage of the disorder comparing the two sides;[3,6,7] (5) girls affected twice as frequently as boys; (6) normal or slight retardation of closure of the epiphysis but persistent deviation and shortening of the diaphysis of the terminal phalanx with virtual return to normal bony architecture;[5,9] (7) no other pathologic changes of the skeleton; and (8) no familial occurrence.

The question of the nature of this disorder has so far remained open to speculation. In our Case 1 we did a biopsy of one of the affected fingers, at which time it was noted that there was a definite lysis between the diaphysis and epiphysis of the terminal phalanx. The biopsy specimen is presently being reviewed by different authorities on bone pathology and will be reported upon in a later communication.

There appears to be evidence that this is a lesion compatible with aseptic necrosis. The observation of an epiphysiolysis, however, presents a new possibility on the etiology of this rare disorder. In the lateral view of the fourth and fifth fingers of the

left hand in our Case 1 (Fig. 6, *A* and *B*), the separation of the diaphysis is clearly apparent. The epiphysis itself is in a normal position but is markedly thicker than normal. Again, the lack of a well defined metaphysis is noticed, together with the absence of normal trabeculation and poor structural formation of the cortex. We emphasize this point particularly since Schmid[5] proposed the term "juvenile osteomalacia of the terminal phalanx of the small fingers" to describe this disorder. We have not been able to obtain any evidence of an osteomalacic process and feel that this disease primarily affects the formation of the metaphyses of the terminal phalanges of the fifth fingers.

As a secondary mechanism, dominance of the flexor digitorum profundus over the extensor muscles displaces the diaphysis in the described manner. Subsequent to epiphyseal lysis there is a sudden aggravation of the abnormal position of the terminal phalanx. The condition then stabilizes in this abnormal anatomic relationship with ultimate fusion, shortening and retrabeculation of the affected terminal phalanx.

Based on the evidence mentioned and awaiting the outcome of the analyses of the pathologic specimen, we propose, for the time being, to refer to this disorder, using the original descriptive term of Kirner,[3] as "bilateral incurving of the terminal phalanges of the fifth fingers."

SUMMARY

A description of 2 cases of bilateral incurving of the terminal phalanges of the fifth fingers is presented. This rare disorder affects adolescents from eight to fourteen years old, and girls twice as frequently as boys. The finding of a true epiphysiolysis, confirmed at operation, is reported for the first time. Available data on the natural course of this disorder are reviewed. There is ultimate bony union of the slipped elements in their abnormal anatomic relationship. No therapy has been proposed up to the present time.

H. J. Kaufmann, M.D.
Kinderspital
Basel, Switzerland

We express our indebtedness to Dr. E. B. D. Neuhauser for permission to include Case II and our gratitude for his help and criticism in the preparation of this paper.

REFERENCES

1. Brailsford, J. F. The Radiology of Bones and Joints. Fourth edition. Williams & Wilkins, Baltimore, 1948, p. 55 and 58.
2. Hippe, H. Krallenförmige Verkrümmung der Endglieder beider Kleinfinger. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1953, *78*, 745.
3. Kirner, J. Doppelseitige Verkrümmungen des Kleinfingerendgliedes als selbständiges Krankheitsbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, *36*, 804–806; referred to in Köhler, A., and Zimmer, E. A. Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology. English translation by Case, J. T. Grune & Stratton, New York, 1956, p. 41.
4. Mercer, W. Orthopaedic Surgery. Third edition. Edward Arnold & Company, London, 1944, p. 89.
5. Schmid, F. Eine juvenile Osteomalazie der Kleinfingerendphalange. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1957, *86*, 766–770.
6. Schmid, F., and Moll, H. Atlas der Normalen und Pathologischen Handskelettentwicklung. Springer-Verlag, West Berlin, 1960, pp. 53–55.
7. Thomas, A. R. New dystrophy of fifth finger. *Lancet*, 1936, *1*, 1412–1413.
8. Tomesku, I. Kongenitale Deviationen der Phalangen (Angeborene Contracturen der Finger und Klinodaktylien). *Arch. f. Orthop.*, 1928, *26*, 126–137.
9. Wilson, J. N. Dystrophy of fifth finger; report of four cases. *J. Bone & Joint Surg.*, 1952, *34B*, 236–239.

SEPARATE CENTERS OF OSSIFICATION OF THE TIP OF THE INTERNAL MALLEOLUS

By SAMUEL SELBY, M.R.C.S. (ENG.), *Fels Research Institute*
YELLOW SPRINGS, OHIO

SEPARATE centers of ossification of the tip of the internal malleolus have been described by Fairbank,[4] Mouchet,[8] and Den Hoed.[3] Bircher[1] has described an os subtibiale, which is without doubt a nonunion of a separate center of ossification at the tip of the internal malleolus. Mouchet[8] reported the findings in a single child. Lapidus[7] stated in *The Journal of Bone and Joint Surgery*, "Ruckensteiner demonstrated the presence of the distal tibial epiphysis as early as the seventh month of life, in any event nearly always before the second year. According to him, the distal tibial epiphysis develops in transverse direction to the long axis of the limb. As the ossification of the epiphysis reaches the medial lower edge of the tibial diaphysis, the ossification of the epiphysis continues medialward and downward, forming the medial malleolus. The latter takes place only at the age of eight or nine years. At about this time an inconstant independent center of ossification may occasionally be formed at the tip of the medial malleolus. This center seems to unite soon after its appearance. One case was seen by Ruckensteiner." The incidence of this lesion was not specifically investigated by any of the mentioned authors. Den Hoed,[3] in studying the roentgenograms of 150 normal children for ossification development, found 21 cases or 10.7 per cent in which there were separate centers of ossification at the tip of the internal malleolus. Of these, 14 were boys and 7 were girls. He contrasted his findings with those of Fairbank[4] who stated that it occurred more commonly in girls.

Köhler and Zimmer[6] in their book state that the separate center of ossification is common between the ages of eight and nine; however, the incidence, sex distribution or the age of complete ossification of the tibial tip are not given. Caffey[2] states, "In a small percentage of healthy children who have no sign of disease near the ankles, the medial malleolus of the distal epiphyseal cartilage of the tibia will show a separate ossification center . . . the ossicles may be unilateral or bilateral." He does not mention the specific incidence percentage nor sex distribution.

METHODS AND MATERIAL

The Fels longitudinal study is an ideal method by which to determine the time of appearance of extra centers of ossification based on sex. In the past years the character of the population at the Fels Research Institute has not substantially changed. The socioeconomic distribution consists of a group in moderate circumstances, Caucasian in origin and from a background of farmers, tradesmen and people of the middle and lower-middle classes. Many siblings of those in the early study have become part of the present sample. Children born after 1937 and before 1953 constitute the present study group. These children repeatedly underwent complete pediatric examinations. Anthropologic measurements were made of their height, weight, length, chest, skull, and calf. They were examined roentgenographically at birth, at one month, three months, six months, nine months, one year and thereafter at half-yearly intervals until the age of eighteen. The examination usually consisted of roentgenograms of the skull, teeth and of the extremities, including soft tissue roentgenograms of the latter, and of an anteroposterior roentgenogram of the left lower limb which was always available for comparison. A special posture roentgenogram was also taken of children between the ages of two and one-half to eight and one-half years. These included both lower limbs, enabling

one to detect the age at which the extra centers of ossification of the tip of the internal malleolus appeared, whether or not they were bilateral and often when they united with the main mass of the tibia. If the center had not yet appeared, or had not joined the main mass by the age of eight and one-half years, its progress could be traced on the roentgenograms of the left lower limb.

This series consisted of 151 children from 75 families, 88 boys and 63 girls. There were two sets of triplets and three sets of twins. Fifteen of the boys showed roentgenographic evidence of extra ossification centers of the tip of the internal malleolus. The earliest age at which these appeared in the boys was six years, the latest age was eleven. The earliest age at which the centers joined the main mass was six months, the latest twelve years. The boy in whom the ossification center appeared at eleven years and joined at twelve matured slowly (Greulich and Pyle[5]). Of the 63 girls, 30 had extra centers of ossification. Twenty-seven of these had roentgen findings of bilateral centers. Three were found to have extra centers of ossification on the roentgenograms of the left limb since they did not fall in the group which had special posture roentgenograms. The earliest age at which the extra centers appeared in the girls was six and one-half years and the latest age was nine years. The latest age at which the ossification center united with the main mass was eleven and one-half years in a girl in whom the extra tibial center first appeared at nine; she, too, was a late maturer[5] and also a severe asthmatic. The shortest period in which the extra ossification center joined the main mass after its appearance in both girls and boys was six months. Posture roentgenograms taken at eight and one-half years showed that only 4 of the 15 boys had bilateral ossification centers; two were brothers. Table 1 shows the age of appearance and union of the extra centers to the main malleolar mass. The mean age of appearance in girls was 7.65 years; the mean age of union was 8.7 years. The median length of time in which union occurred was 1.14 years. In boys the mean age of appearance was 8.76 years and of union 10.8 years. The median length of time in which union occurred was 1.34 years.

Of the boys, 2 sets of brothers had two extra ossification centers; in the girls 6 sets of sisters had extra centers of ossiffication. One of the group of sisters had a brother who also had an extra center. This was the only family in which the extra centers ap-

TABLE I

AGE OF APPEARANCE OF EXTRA CENTERS

Age (yr.)	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0	10.5	11.0	Combined Total
Boys	1	—	1	1	1	2	5	1	2	—	1	15
Girls	—	6	1	11	3	6	3	—	—	—	—	30
Total	1	6	2	12	4	8	8	1	2	—	1	45

AGE AT WHICH THE EXTRA CENTER JOINED TIBIA

Age (yr.)	6.0	7.5	8.0	8.5	9.0	9.5	10.0	10.5	11.0	11.5	12.0	Combined Total
Boys		1	—	1	1	—	4	—	4	—	1	12
Girls		3	3	10	6	2	4	—	—	1	—	29
Total		4	3	11	7	2	8	—	4	1	1	41

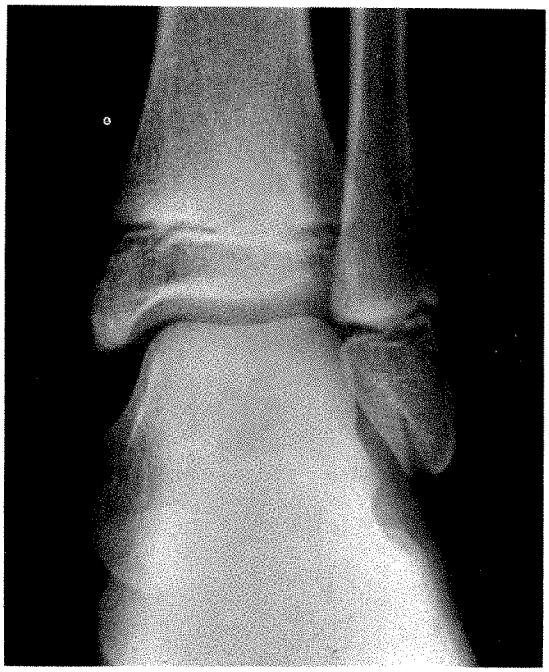


FIG. 1. Female, aged eight years. Roentgenogram shows beginning downgrade of tip.

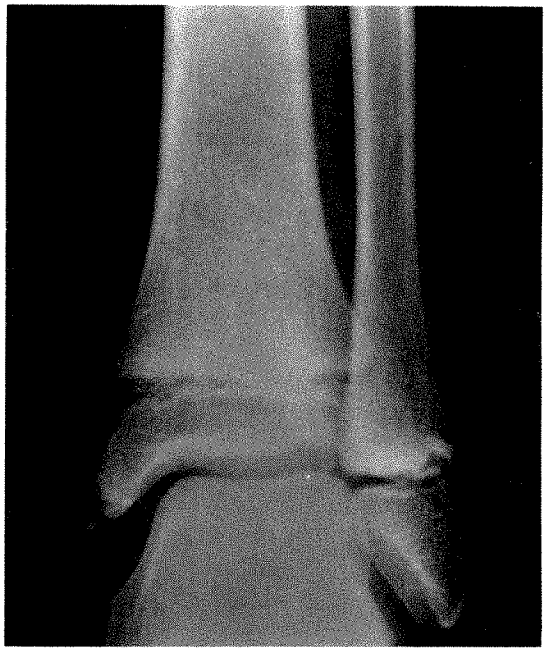


FIG. 2. Female, aged eight and one-half years. Roentgenogram shows a separate ossification center.

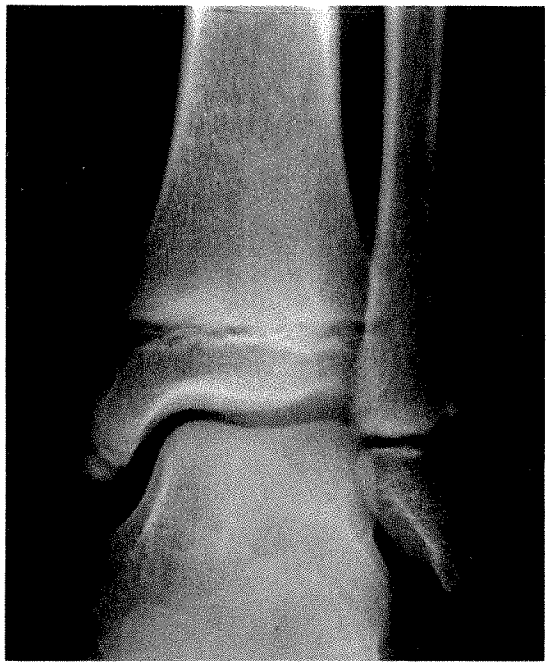


FIG. 3. Female, aged nine years. Roentgenogram shows engulfing of separate ossification center, giving the appearance of an os subtibiale.



FIG. 4. Female, aged eight and one-half years. Roentgenogram shows the downgrowth of the main mass and two separate ossification centers.

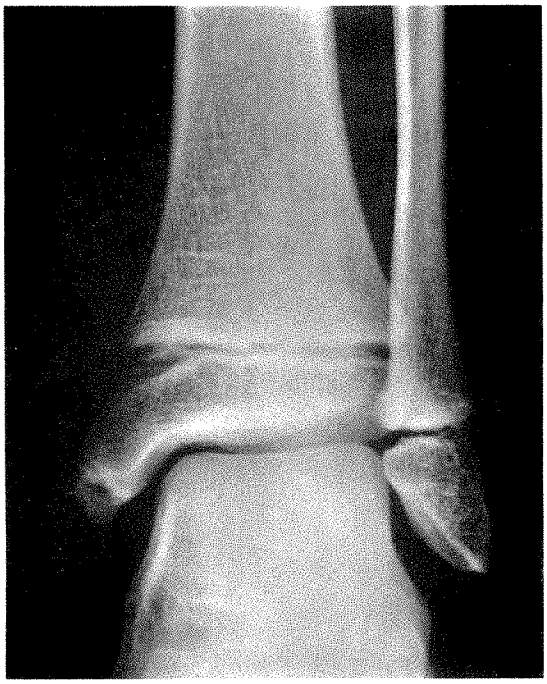


Fig. 5. Female, aged nine years. Roentgenogram shows 4 ossification centers outlining the tip.

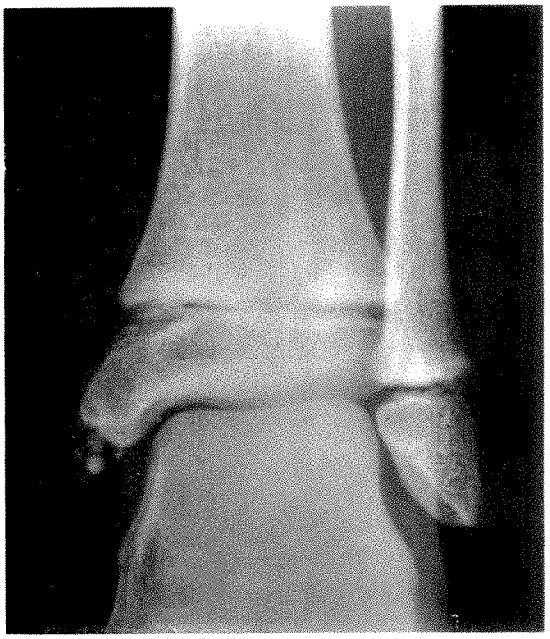


Fig. 6. Female, aged nine and one-half years. Roentgenogram shows formation of the tip with spherule like areas.

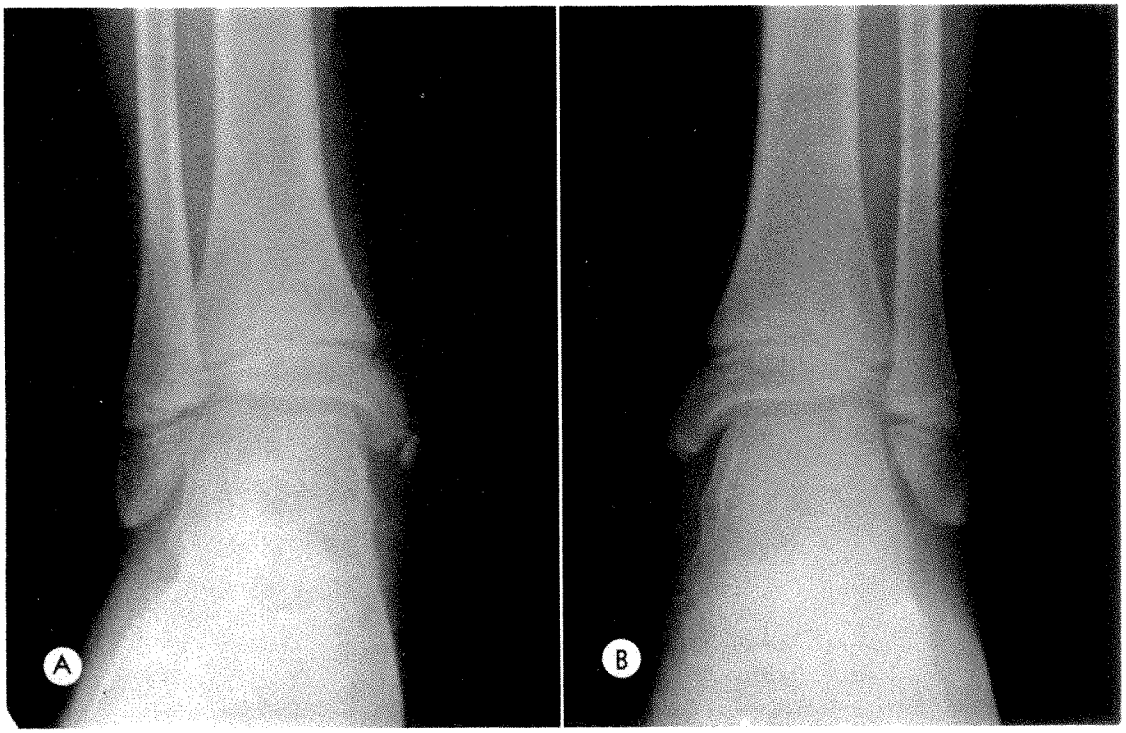


Fig. 7. (*A* and *B*) Female, aged seven and one-half years. Roentgenograms show the formation of the tip of the internal malleolus in an early maturer.

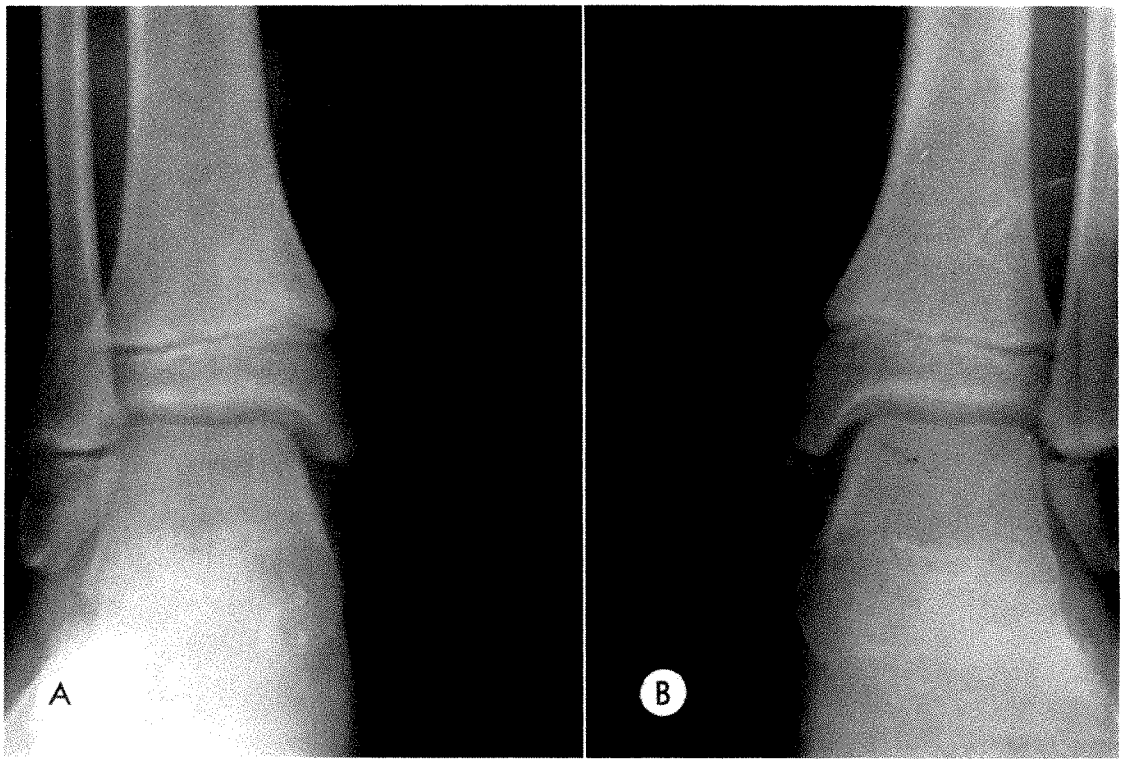


FIG. 8. (*A* and *B*) Female, aged seven and one-half years. Roentgenograms showing that the tip of the malleolus of the right extremity formed ahead of the one of the left extremity.

peared uniformly in all 3 children. Extra ossification centers were regarded as such only when there was a definite radiolucent boundary between the extra center and the main mass of the lower tibial epiphysis (Fig. 1 and 2). This was the defining boundary which Bircher[1] used in his description of the os subtibiale (Fig. 3). It was found necessary to do this because the rapid downgrowth of calcification, often comma-shaped, could be confused with an extra center. Frequently, there was downgrowing calcification engulfing and surrounding the extra centers which made them look like a conjoined group of spherules (Fig. 4), or multiple centers (Fig. 5). Sometimes the center began as a small millet seed (or it could be larger), then from time to time appeared as two to three discrete small centers (Fig. 6). At times (Fig. 7, *A* and *B*) a separate center of the tibia would appear on the right side before one appeared on the left, or an ossification center on one side might form a tip of the tibia without an extra center (Fig. 8, *A* and *B* and 9, *A* and *B*).

There does not seem to be a uniform pattern in the formation of separate centers of ossification; however, they do tend to be bilateral in females.

CONCLUSIONS

Extra centers of ossification at the tip of the internal malleolus are found more frequently in females than in males. They have been noted in 47 per cent of females. This appears to be a hereditary trait.[9] Pryor[10] observed also that in females the skeletal ossification occurs at an earlier age.

9A Langland Gardens
London N.W.3, England

I am indebted to Christabel Rohmann for statistical help and to Lois Conklin for her patience in typing this article.

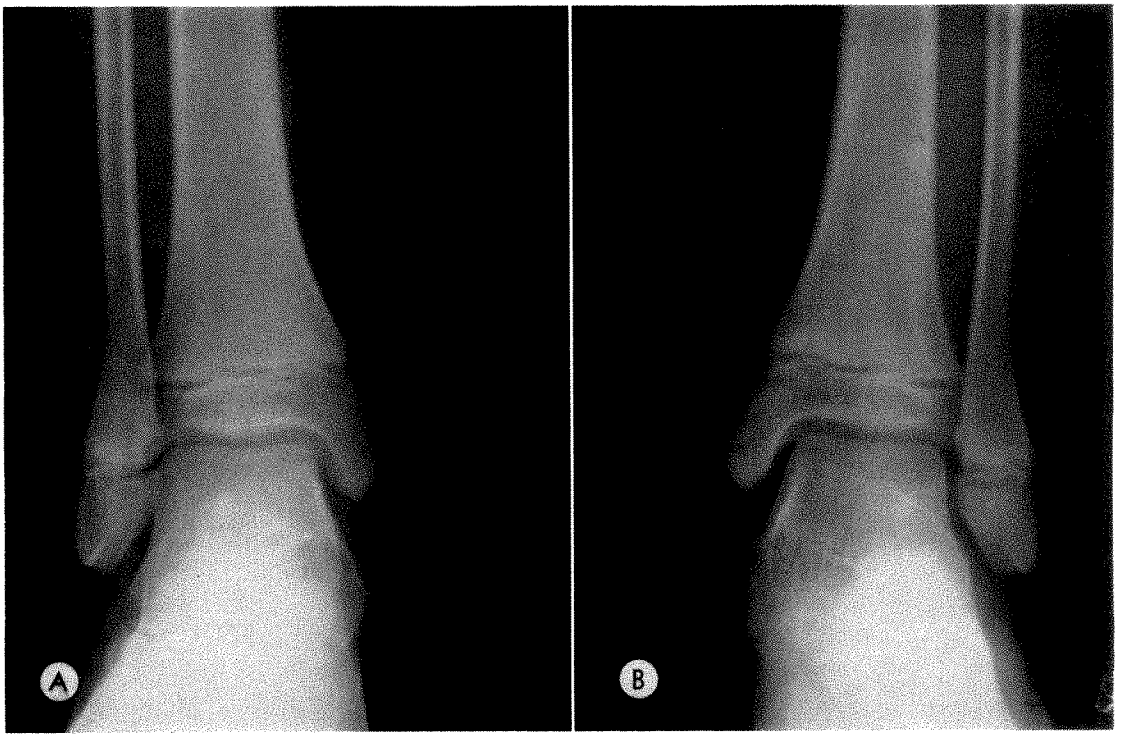


Fig. 9. (*A* and *B*) Female, aged eight and one-half years. Roentgenograms show that the tips of the malleolus are formed in both extremities; however, the right is more advanced than the left. Variations in radiolucency can be seen on the left extremity.

REFERENCES

1. Bircher, E. Os subtibiale. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1918–1919, *26*, 85–88.
2. Caffey, J. Pediatric X-ray Diagnosis. Third edition. The Year Book Publishers, Inc., Chicago, 1956, p. 722.
3. Den Hoed, D. A. Separate centre of ossification for tip of internal malleolus. *Brit. J. Radiol.*, 1925, *30*, 67–68.
4. Fairbank, H. A. T. Separate centre of ossification for tip of internal malleolus. *Arch. Radiol. & Electrotherapy*, 1923, *27*, 238–241.
5. Greulich, W. W., and Pyle, S. I. Radiographic Atlas of Skeletal Development of the Hand and Wrist. Second edition. Stanford University Press, Stanford, 1959.
6. Köhler, A., and Zimmer, E. A. Grenzen des Normalen und Anfänge des Pathologischen im Röntgenbilde des Skelettes. Ninth edition. Georg Thieme Verlag, Stuttgart, 1953, p. 536.
7. Lapidus, P. W. Os subtibiale; inconstant bone over tip of medial malleolus. *J. Bone & Joint Surg.*, 1933, *15*, 766–771.
8. Mouchet, A. Point d'ossification du sommet de la malléole tibiale. *Bull. et Mém. Soc. de Chir. de Paris*, 1923, *49*, 709–799.
9. Pryor, J. W. Hereditary nature of variation in ossification of bones. *Anat. Rec.*, 1907, *1*, 84–88.
10. Pryor, J. W. Differences in time of development of centers of ossification in male and female skeleton. *Anat. Rec.*, 1923, *25*, 257–268.

UNUSUAL BONE TUMORS AFTER ROENTGEN THERAPY OF CHILDREN*

TWO CASE REPORTS

By JONATHAN COHEN, M.D., *and* GIULIO J. D'ANGIO, M.D.
BOSTON, MASSACHUSETTS

POSTIRRADIATION tumors of bone, both malignant and benign, have occurred infrequently following roentgen therapy which included exposure of parts of the normal skeleton (Tables IA and IB).[26] The appearance of two unusual tumors of bone in a selected series of 247 children who received roentgen-ray therapy at The Children's Hospital Medical Center of Boston has important implications regarding the late effects of radiation in children. The tumors are of interest because of their histologic features, which are distinctly different from any postirradiation tumors of bone previously recorded and which present some difficulty in classification. In the series, the 2 cases represent the only instances of neoplasms, excluding exostoses, which occurred in normal bone after roentgen therapy. In the literature, the most frequent malignancies induced in bone by irradiation have been osteogenic sarcomas. Rarely, chondrosarcomas, giant cell sarcomas, and unclassified sarcomas have appeared. The reported benign postirradiation neoplasma have been osteochondromas.

MATERIAL

The series of children under discussion was selected from the total of 8,321 who were referred to the Radiotherapy Section of The Children's Hospital Medical Center of Boston from January, 1938 to December, 1957. With rare exceptions, all were under thirteen years of age.

They could be classified in two main groups. In the first, 7,423 had benign processes, *e.g.*, hemangiomas, nasopharyngeal lymphoid hyperplasia, keloids, etc. Not all these children were treated. When roentgen irradiation was given, the estimated doses to bone were very rarely more than 1,000 r and, in the majority of patients, less than 500 r. These children, therefore, received lower doses than might be expected to cause bone tumors. They have been followed for many years, either in hospital clinics or by referring physicians. To the best of our knowledge, no instance of postirradiation bone tumor has occurred. Consequently, this group is excluded from further consideration here.

The second group includes 898 children who received more intensive and protracted courses of treatment, the majority for malignant processes, a smaller number for Hand-Schüller-Christian disease or its variants, and a few for aneurysmal bone cysts, nasopharyngeal fibromas, etc. Follow-up data on all but 35 of these children are available. Two hundred and forty-seven have survived for two years or longer after roentgen therapy (Table II). All had treatment administered to portals that included at least one normal bone. The bones most commonly irradiated were the vertebrae, the ribs, and the skull. Of the 247 patients, 207 (84 per cent) had doses of 1,000 r or more delivered to bone.

REPORT OF CASES

CASE I. R.O. was first seen on January 14, 1943 when he was ten months of age. His chief complaint was a mass in the left flank. He was admitted to the hospital where a clinical diag-

* From the Departments of Orthopedics, Radiology, and Pathology, The Children's Cancer Research Foundation; and the Division of Laboratories and Research, The Children's Hospital Medical Center; and the Departments of Orthopedics and Radiology, Harvard Medical School, Boston, Massachusetts.

This investigation was supported in part by grants from the National Institutes of Health, USPHS #CY3335.

TABLE 1A

REPORTED BONE TUMORS ARISING IN *NORMAL BONE* AFTER ROENTGEN THERAPY DURING CHILDHOOD

No.	Age When Treated (yr.)	Sex	Latent Period (yr.)	Primary Disease	Bone Affected	Estimated Dose Delivered to Bone	Tumor Type	Reported by
1	11	M	3	Tuberculous knee	Femur	Not stated	Osteogenic sarcoma	Beck[1]
2	6	M	6	Tuberculous knee	Femur	Not stated	Giant cell sarcoma	Marsch[23]
3	9	M	8	Tuberculous knee	Femur	Not stated	Chondrosarcoma?	Marsch[23]
4	7	M	8	Tuberculous knee	Femur and tibia	Not stated	Giant cell sarcoma	Jaruslawsky[14]
5	8	F	3	Tuberculous knee	Femur	Not stated	Osteogenic sarcoma	Küttner[16]
6	5	F	7	Tuberculous knee	Femur	Not stated	Osteosarcoma	Denks[6]
7	11	M	8	Tuberculous knee	Tibia	Not stated	Osteogenic sarcoma	Denks[6]
8	11	F	5	Tuberculous knee	Femur	Not stated	Chondrosarcoma?	Gruca[10]
9	4	M	12	Soft tissue sarcoma	Femur	7,700 r	Osteosarcoma	Cade[2]
10	1	F	10	Retinoblastoma	Frontal	?	Osteosarcoma	Skolnik *et al.*[27]
11	2	F	8	Retinoblastoma	Ethmoid or maxilla	>12,000 r	Osteosarcoma	Tebbet and Vickery[28]
12	12½	M	3	Poliomyelitis	Femur	6,800 r	Osteosarcoma	DeYoung[7]
13	?	?	20	Wilms's tumor	Rib	?	Osteosarcoma	Lichtenstein[19]
14	1 mo.	F	13½	Cystic hygroma	Clavicle	?	Osteosarcoma	Cruz *et al*[5]
15	2	F	6	Retinoblastoma	Ethmoid	25,000 r	Osteosarcoma	Cahan *et al.*[3]

TABLE 1B

REPORTED BONE TUMORS ARISING IN *ABNORMAL BONE* AFTER ROENTGEN THERAPY DURING CHILDHOOD

No.	Age When Treated (yr.)	Sex	Latent Period (yr.)	Primary Disease	Bone Affected	Estimated Dose Delivered to Bone	Tumor Type	Reported by
1	9	M	7	Granuloma	Maxilla	175 r+1,200 mg./hr. Ra	Osteogenic sarcoma	Sabanas *et al.*[26]
2	10	M	13	Bone cyst	Humerus	6,000–9,000 r	Osteogenic sarcoma	Cahan *et al.*[3]
3	7	M	17	Bone cyst	Femur	1,550 r	Chondrosarcoma	Cahan *et al.*[3]
4	12	M	9	Bone cyst	Humerus	5,580 r	Osteogenic sarcoma	Cahan *et al.*[3]
5	13	F	13	"Cloudiness of antrum"	Maxilla	3,600 r	Osteogenic sarcoma	Cahan *et al.*[3]
6	1	F	7	Bone cyst	Tibia	?	Chondrosarcoma	Francisco *et al.*[8]
7	5	M	7	Bone cyst	Humerus	?	Osteogenic sarcoma	Cruz *et al.*[5]
8	12	M	5	Bone cyst	Humerus	?	Fibrosarcoma	Jaffe[12]
9	9	F	7	Aneurysmal bone cyst	Femur	2,600 r	Fibrosarcoma	Lichtenstein[20]

TABLE II

SELECTED SERIES OF CHILDREN TREATED AT THE CHILDREN'S HOSPITAL MEDICAL CENTER (JANUARY, 1938 THROUGH DECEMBER, 1957) WHO SURVIVED TWO YEARS OR LONGER

Diagnosis	100–499 r	500–900 r	1,000–1,900 r	2,000–2,900 r	3,000–3,900 r	4,000–4,900 r	5,000–5,900 r	6,000–6,900 r	7,000+ r	Total No. of Cases
Central Nervous System Tumors	1	2	8	19	16	9	3	3	2	63
Wilms's Tumor	0	0	9	21	18	7	1	1	0	57
Neuroblastoma	1	1	11	17	6	1	1	0	0	38
Hand-Schüller-Christian Disease	3	22	6	0	0	0	0	0	0	31
Lymphoma	0	2	11	5	2	0	0	1	0	21
Others	4	4	11	8	6	2	0	1	1	37
Total	9	31	56	70	48	19	5	6	3	247

nosis of Wilms's tumor (embryoma) of the left kidney was made. The family history was noncontributory, as was the child's gestational, neonatal, and developmental history.

Physical examination elicited normal findings except for a large, firm, smooth mass in the left flank. The tumor extended from the costal margin to the iliac crest and from the lateral abdominal wall to the midline.

Routine laboratory studies were within normal limits, except for nonprotein nitrogen of 45.2 mg./100 cc. Roentgenograms of the chest and long bones were negative. An intravenous pyelogram showed a large, poorly circumscribed soft tissue mass on the left, without abnormality of the right kidney and ureter.

On January 15, 1943 a laparotomy was performed. The tumor had infiltrated the posterior peritoneal wall, adjacent lymph nodes, and the renal vein. It was irregularly lobulated and arose from the lower pole of the kidney. It could not be completely removed and a left nephrectomy was done. Histologically, the tumor was composed of large sheets and islands of neoplastic cells. There was invasion of surrounding tissues, including the vessels. The pathologic diagnosis was embryoma.

Following the nephrectomy, roentgen therapy was administered with the following technical factors: 200 kvp.; 0.5 mm. Cu and 1 mm. Al added filtration (half value layer of 1.05 mm. Cu); 10 ma.; 50 cm. target skin distance. The free air dose rate at 50 cm. was 28 r per minute. (The doses which follow are all expressed as measured in air.) Daily, except Sundays, 200 r was administered to alternating anterior and posterior portals. The fields, which measured 9.5×13 cm. extended from the right paravertebral plane to the left flank and from the level of D6 to S2. Fifteen treatments in nineteen days (January 16 to February 3, 1943) were given, with total doses of 1,400 r and 1,600 r to the anterior and posterior portals, respectively.

The child did well and was readmitted on June 6, 1944, at which time no recurrence of the tumor was evident. Roentgen therapy was again given, the technical factors being the same as those listed above. Opposing anterior and posterior ports were used, this time measuring 9×12 cm. After an initial treatment of 200 r on June 8, 1944, the daily dose was changed to 300 r, and seven additional treatments in eight days were delivered. The course terminated on June 16, 1944. Thus, a total dose of 1,100 r was given to the anterior portal and 1,200 r to the posterior portal.

Calculations based on the above data were made for the total doses delivered to the left posterior rib cage (1 cm. below the skin) and to the tumor bed at the midplane. Standard depth dose tables[9] were employed and no corrections were made for the lack of homogeneity of the irradiated tissues, uneven distribution of dose across the field, etc. The figures which follow are approximations and are not meant to be precise assessments of the doses delivered. Thus, 2,566 r was received by the posterior portions of the ribs in the first course of roentgen therapy, while the tumor dose was 1,950 r as calculated at the midplane. The corresponding dosages during the second course were, 1,697 r and 1,357 r, totalling 4,263 r and 3,307 r to ribs and tumor, respectively. Using an appropriate conversion factor, the estimated rib dose in rads was 7,503.[24]

Following this therapy the boy was seen at intervals and continued to do well. When he was fifteen years old, thirteen years after the completion or roentgen therapy, he first felt a lump in his chest which was slightly tender and painful. Two weeks thereafter he was readmitted to the hospital. At this time, physical examination revealed a well-healed left rectus incision and no other abnormal findings except for a firm tender mass 3 cm. in diameter, associated with the eleventh left rib. Routine laboratory examinations were normal except for occasional hyaline casts in the urine.

Roentgen examination (Fig. 1) showed an irregularity of the bodies of the lumbar vertebrae, without erosion or destruction. The left ilium was small and underdeveloped. A mild scoliosis in the thoracolumbar area was noted. The left eleventh rib showed an irregular architecture and expanded contour, with mottled density and punctate calcific deposits within a surrounding soft tissue mass.

Operation on September 4, 1957 disclosed a firm mass, 4×3×5 cm., involving the eleventh rib. The cut surface showed several segments of paper-thin bony cortex which was entirely absent over the medial aspect. The tumor, a solid, nodular, white, and translucent mass, extended through the pleura, which was not infiltrated. An *en bloc* excision was performed, with the removal of the tenth, eleventh, and twelfth ribs by disarticulation from the vertebrae. The associated soft tissues, including intercostal

muscles, blood vessels and nerves, were also excised.

Microscopically (Fig. 2; and 3, *A–C*), the tumor was found to have broken through a thin bony cortex at a number of places and to have extended into the adjacent fibrous tissue but not into the muscle. Nodules of abnormal cartilage, separated by thin fibrous septa, comprised most of the tumor. The centers of the nodules were often composed of mature cartilage with fewer cells and more matrix than in the periphery. Myxoid tissue was present in abundance. The cartilage cells were variable in shape and size and many had atypical nuclei and occupied large lacunae, some of which contained two and three cells. No gaint cells, areas of calcification, or bone formation were noted. In the periphery

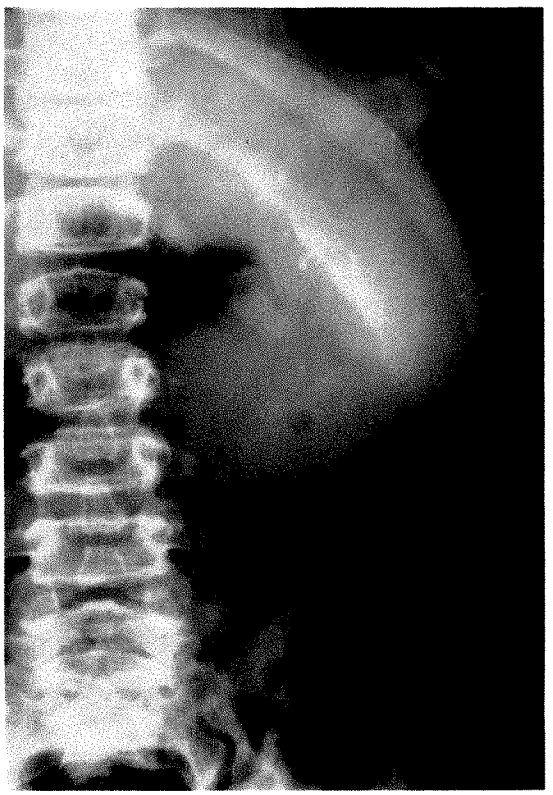


Fig. 1. Case 1. A fifteen year old boy with a bone tumor developing thirteen years after roentgen therapy of the left hemiabdomen. The left eleventh rib, normal at the time of irradiation, now shows expansion and irregularity of outline. There is disruption of the normal bony architecture. Mottled calcification is present within an associated soft tissue mass. Postirradiation abnormalities in the contour and size of the left ilium and of the dorsal and lumbar vertebrae are visible.



Fig. 2. Case 1. Low power magnification of a cross section of the tumor. The bony portion of the rib is surrounded by tumor the lobulation of which is evident. The smaller, darker-stained lobules contain calcified cartilage. Small plaques of bone are seen in the left upper and right lower portion. The pleura is at the inferior edge of the mass.

of some nodules, rows of cells were present which resembled fibroblasts separated by cords of atypical nonfibrillar matrix.

Postoperatively the boy did well and two years later showed no evidence of tumor. However, the scoliosis (apex at D10) showed a marked increase and at that time measured 15° in recumbency and 25° in the upright position. No symptoms were present.

Case II.* B.H., a Negro girl, first complained of abdominal swelling and vomiting in April, 1954, when she was three years old. At another hospital, physical examination revealed an abdominal mass which was diagnosed as Wilms's tumor. It was found to infiltrate the liver and, during its removal, it was excised "flush with the liver" but abdominal spillage of tumor cells occurred. Her postoperative course was stormy and was complicated by hemorrhage, shock, suture of a partially divided common iliac artery, and adrenal insufficiency requiring prolonged ACTH and cortisone therapy.

Postoperatively, roentgen therapy was given to the right upper abdomen to anterior, lateral, and posterior portals, each measuring 10×15 cm. Air doses of 1,300 r, 1,200 r, and 1,000 r, respectively, were given to these three portals from April 8 to 27, 1954. The technical factors

* Details regarding the early roentgen treatment given this patient are reported through the kind cooperation of Dr. B. B. Alicandri, Mercy Hospital, Springfield, Massachusetts.

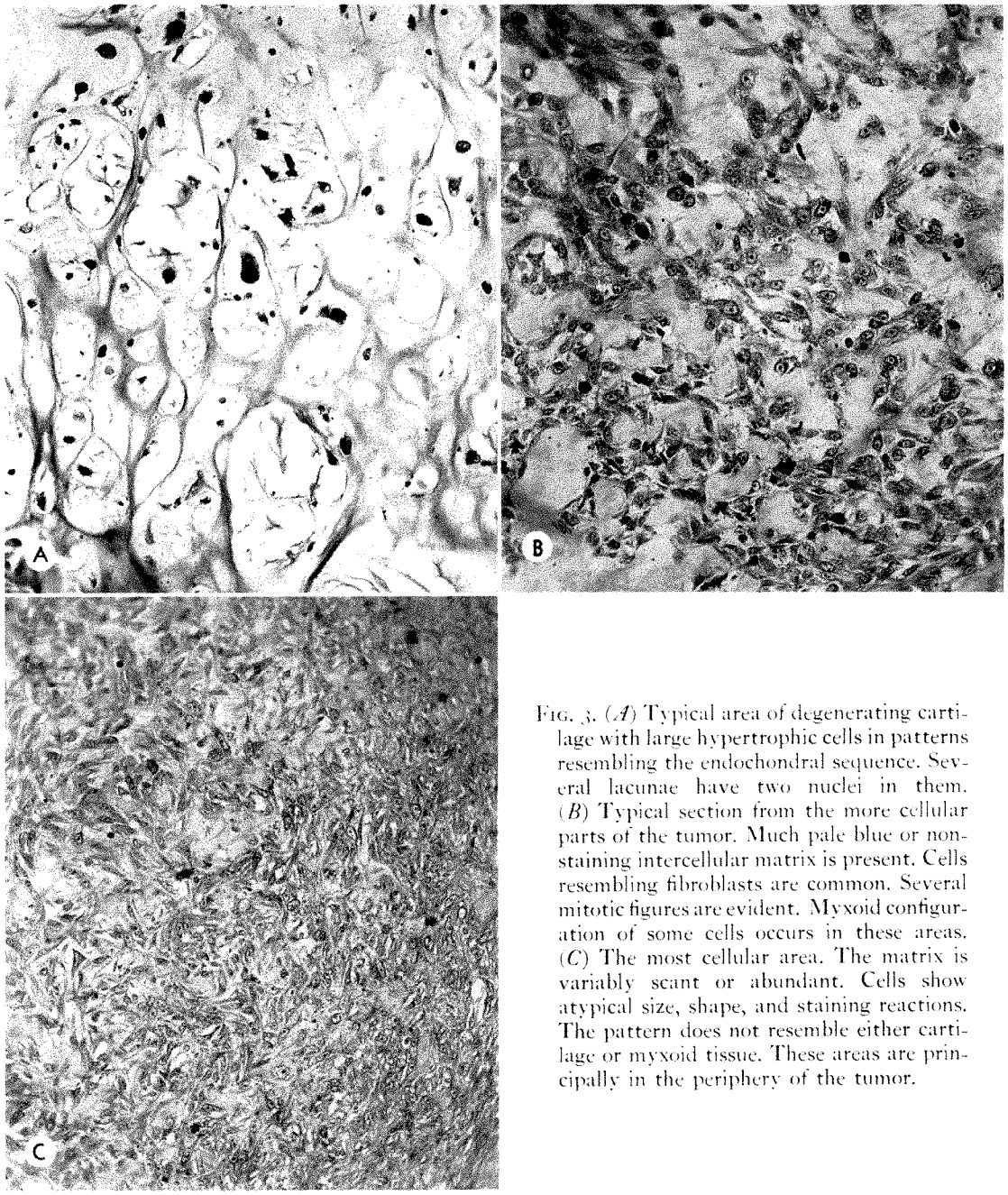


FIG. 3. (*A*) Typical area of degenerating cartilage with large hypertrophic cells in patterns resembling the endochondral sequence. Several lacunae have two nuclei in them. (*B*) Typical section from the more cellular parts of the tumor. Much pale blue or nonstaining intercellular matrix is present. Cells resembling fibroblasts are common. Several mitotic figures are evident. Myxoid configuration of some cells occurs in these areas. (*C*) The most cellular area. The matrix is variably scant or abundant. Cells show atypical size, shape, and staining reactions. The pattern does not resemble either cartilage or myxoid tissue. These areas are principally in the periphery of the tumor.

employed yielded a half value layer of 1 mm. of Cu; the target skin distance was 50 cm. From June 9 to 19, 1954, an additional 1,200 r in air was given to a posterior portal directed to the right upper renal fossa through a 10×10 cm. field. At the same time, treatment was directed to a left hilar nodule, employing 10×10 cm. opposing thoracic portals. An air dose of 660 r was given to the anterior chest and 1,200 r to the posterior. The depth dose was calculated at the level of the left rib cage posteriorly (1 cm. below the skin) and in the midplane in the same manner as in Case 1. The total estimated dose to the midplane was 1,246 r and to the posterior ribs, 1,800 r (3,168 rads).

After this therapy the liver began to enlarge and on August 11, 1954 the patient was first referred to this hospital. At that time physical ex-

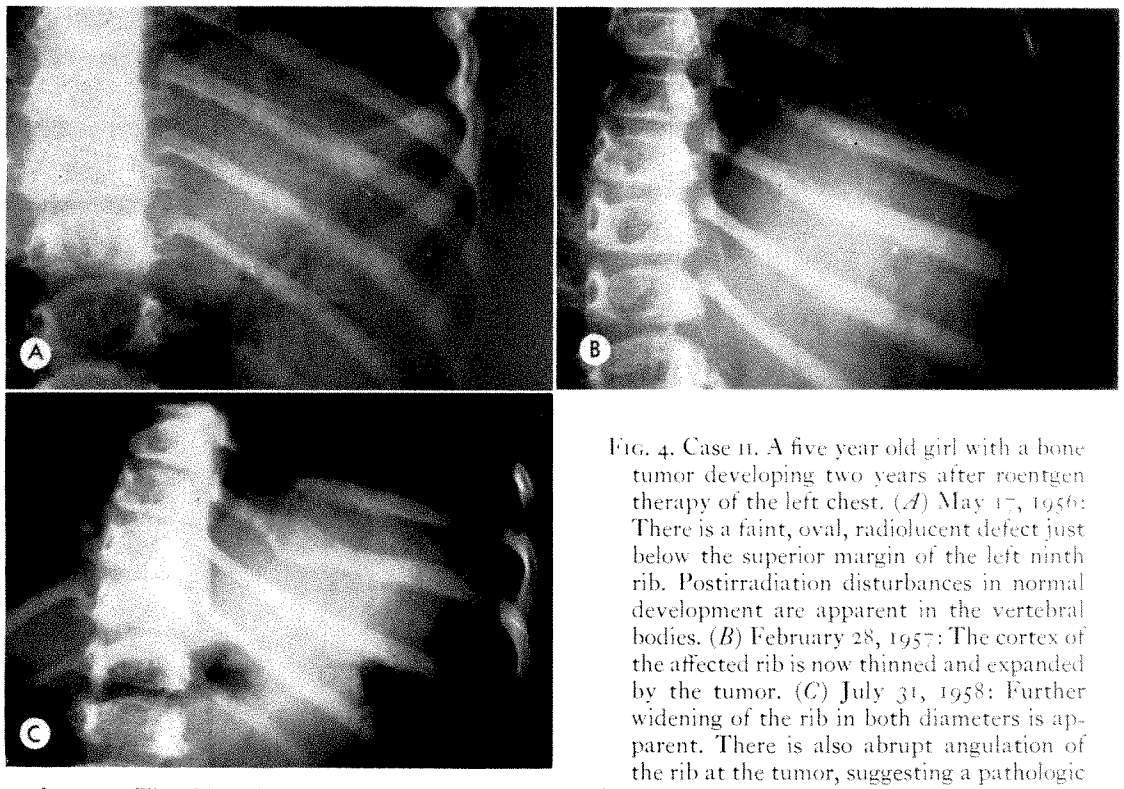


FIG. 4. Case II. A five year old girl with a bone tumor developing two years after roentgen therapy of the left chest. (*A*) May 17, 1956: There is a faint, oval, radiolucent defect just below the superior margin of the left ninth rib. Postirradiation disturbances in normal development are apparent in the vertebral bodies. (*B*) February 28, 1957: The cortex of the affected rib is now thinned and expanded by the tumor. (*C*) July 31, 1958: Further widening of the rib in both diameters is apparent. There is also abrupt angulation of the rib at the tumor, suggesting a pathologic fracture. The thinned cortex is intact along both surfaces and the margins of the lesion are sharp, without infiltration of the adjoining rib shaft. No soft tissue mass is visible.

amination showed a poorly developed girl whose principal physical findings indicated a fading radiodermatitis of the abdominal wall and a slight enlargement of the liver. The laboratory findings were within normal limits and the roentgenographic survey of the chest and skeleton showed only an increase in lung markings which was interpreted as radiation fibrosis.

Treatment with a number of antitumor chemotherapeutic agents was then begun and continued intermittently until the present. The child did well until November, 1954, at which time sudden flaccid paralysis of the right lower extremity without sensory loss was noted. She was again hospitalized and a presumptive diagnosis of anterior poliomyelitis was made after myelograms and other pertinent tests did not reveal any other explanation for the paralysis. The neurologic findings have persisted to the present; otherwise, the patient has been asymptomatic, albeit underweight and slow in developing.

In September, 1958, a lesion of the ninth left rib which expanded the cortex was discovered on routine roentgenography. Retrospectively, the lesion could be seen as early as May 17, 1956, at which time it was merely a juxtacortical radiolucency, 2 cm. in length, within the rib shaft and evident posteriorly (Fig. 4*A*). Serial roentgenograms showed gradual growth of the lesion, which had begun to expand the cortex (Fig. 4, *B* and *C*). By September, 1958 it measured 2.0×1.3 cm. and had widened the rib in an irregular fashion. No palpable mass was present.

On September 19, 1958 a segment of the ninth rib was removed. The resection included periosteum and normal bone on both sides. At operation, no invasion of soft tissues by the tumor was noted. A postoperative roentgenogram taken on September 25, 1959 is shown in Figure 5.

Gross examination of the specimen removed at operation revealed a recent fracture through the thin cortex. The cancellous bone of the marrow was replaced by gray, firm, fibrous tissue which, on section (Fig. 6, *A*, *B* and *C*), showed densely packed cells with variable

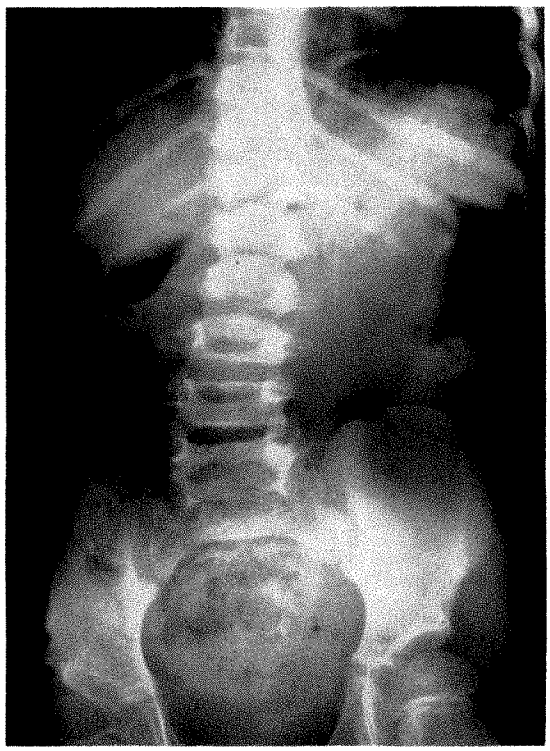


FIG. 5. Case II. September 25, 1959: Postoperative roentgenogram shows that a portion of the left ninth rib posteriorly is absent and there is a small osteochondroma of the right ilium. Postirradiation scoliosis and underdevelopment of the right innominate bone are also present.

amounts of cellular matrix between them. The patterns of the matrix ranged from fairly mature trabecular cancellous bone to primitive fibrillar collections between masses of cells. Foci of giant cells were present. No cartilage, fibrosis, inflammation, or degeneration was present. None of the cells showed atypical nuclear staining, and mitotic figures were infrequent.

DISCUSSION

Only circumstantial evidence supports the contention that radiation has caused neoplasms to develop in cases such as those reported here. Points in favor of this cause-and-effect relationship in the present cases are: (1) the involved ribs showed no abnormality before irradiation. There is no roentgen or histologic evidence to suggest that the tumors arose in pre-existing abnormalities of the bone substance. (2) The dose of radiation was sufficient in both patients to cause growth disturbances in areas immediately adjacent to the involved ribs. (3) The incidence of primary tumor in ribs is infrequent and only a few cases have been reported. It is highly improbable, therefore, that these 2 tumors were primary, spontaneous lesions not associated with irradiation but arising coincidentally in the small group of patients who comprised the selected series. (4) Evidence for the carcinogenic action of irradiation on bone is well documented experimentally and clinically with respect to radium and mesothorium, and experimentally with respect to other bone-seeking radioisotopes. There are only a few reports of the possible carcinogenic potency of local external roentgen irradiation in animals.[17,18,21,22] Perhaps the long latent periods and paucity of postirradiation bone tumors in patients have discouraged experimental studies on this subject. The frequency with which these neoplasms develop in bone is not known with certainty. They must be rare because only 86 cases have been reported. It is noteworthy that 24 of these were in children thirteen years old or younger at the time of irradiation. This frequency in children of over 25 per cent of the reported cases is markedly disproportionate, since children make up only a small fraction of the patients seen in a general radiotherapy center. In 15 of the 24 children listed in Table 1A, neoplasms developed in normal bones included in the field of roentgen therapy. Only 1 of these children received treatment for Wilms' tumor. He developed an osteogenic sarcoma in a rib, the same bone affected in our 2 patients. No other rib tumors developed in the children listed in Tables 1A and 1B.

The relationship between total roentgen dose, dose rate, and latent period, and the development of bone tumors (malignant and benign) is not clearly defined. Jones[15] has published a comprehensive report on the problem of dosage in postirradiation sarcoma. On the basis of his own experience and the experience of others, he supports

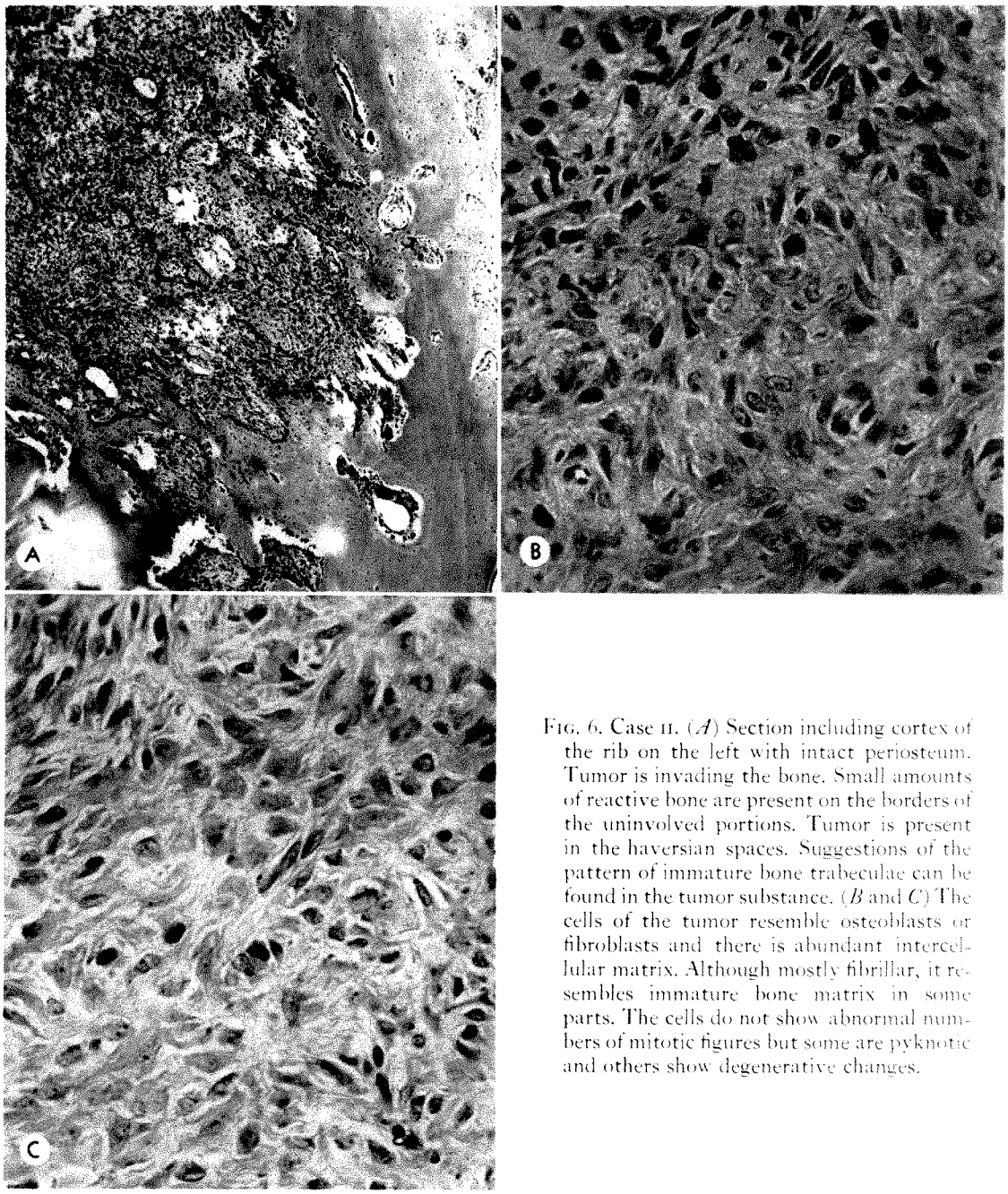


FIG. 6. Case II. (*A*) Section including cortex of the rib on the left with intact periosteum. Tumor is invading the bone. Small amounts of reactive bone are present on the borders of the uninvolved portions. Tumor is present in the haversian spaces. Suggestions of the pattern of immature bone trabeculae can be found in the tumor substance. (*B* and *C*) The cells of the tumor resemble osteoblasts or fibroblasts and there is abundant intercellular matrix. Although mostly fibrillar, it resembles immature bone matrix in some parts. The cells do not show abnormal numbers of mitotic figures but some are pyknotic and others show degenerative changes.

the contention advanced by Cahan *et al.*[3] that a threshold dose of about 3,000 r is necessary for the production of malignant tumors. Two extensive reviews[5,26] and several additional case reports have since appeared. In some patients in whom the dose could be estimated accurately, secondary malignant bone tumors developed after small doses—for example, 1,000 r. More often, however, the level of 3,000 r was exceeded.

The latent period in the 38 cases reviewed by Jones[15] ranged from 3 to 22 years from the end of irradiation, with a mean interval of 8.6 years. The latent period in our second case (two years) is the

shortest hitherto recorded in children (Tables IA and IB), while the latent period in our first patient is well beyond the average reported.

Several reviews on the histology of post-irradiation sarcoma of bone have been published. Sabanas *et al.*,[26] in 1956, summarized the 53 cases recorded in their bibliography and added 17. Most of them had radiation therapy for various types of lesions of bone, and the sarcomas, therefore, arose in pathologic tissue which had been irradiated. Only 3 of their own cases and 10 among those they had reviewed occurred in normal bones which happened to be in the path of radiation administered for adjacent lesions. Three patients developed osteogenic sarcomas, which appeared thirteen, twenty-one, and thirty years after radiation therapy. In the 10 cases which they reviewed, osteogenic sarcomas also predominated, the exceptions being 1 case reported by Copeland[4] (fibrosarcoma superimposed on periosteal osteoma) and 3 reported by Hatcher[11] (chondrosarcoma). Since the study of Sabanas *et al.* was published, 6 additional reports have added 16 cases to the total of bone sarcomas as sequelae to irradiation, administered mostly to adults. Ten of these tumors occurred in previously normal bone tissue. Of these, the 4 cases reported by Cruz *et al.*[5] in 1957 were osteogenic sarcomas, as were 5 of the other cases reported. The tenth was a fibrosarcoma reported by Cade.[2]

From the histologic data in the articles reviewed, only 2, or possibly 3, instances of chondrosarcoma have been recorded subsequent to irradiation of previously normal bone. Cartilage is present in many post-irradiation osteogenic sarcomas. The same is true of the experimental postirradiation sarcomas after administration of radioisotopes. However, cartilage rarely has been the predominant tissue in the clinical cases, as it was in our patient, R. O. (Case 1). His tumor was distinctly different from the others in the literature for which histologic data are presented, including the chondrosarcomas reported. Hatcher's 3 patients showed areas of reactive bone formation and fibrous elements of neoplastic type, in addition to malignant cartilaginous tissue. The tumor in our Case 1 did not contain any elements other than cartilage and myxoid tissue. Although lobulated, the cartilage and myxoid structure was extensive and confluent, a finding not seen in postirradiation osteogenic sarcomas. In those cases, cartilage when present usually was found in small nodules interspersed in the main tissue mass.

There was a wide diversity of opinion on the diagnosis of the tumor in our patient, R. O. The authors and some consultants believed it represented a chondrosarcoma. Others have thought that the tumor was nonmalignant, *i.e.*, either a chondromyxoid fibroma or a chondroma. A diagnosis of chondromyxoid fibroma is usually based on the presence of numerous areas of myxoid tissue in which the cells show some bizarre features. These were occasionally seen in the lesion of R. O. Though the cortex was breached, there was no unequivocal extension into the surrounding muscle, so we cannot exclude the possibility that fibrous tissue still encompassed the tumor. However, the clinical history of rapid enlargement of a painful tender mass argues against the diagnosis of chondromyxoid fibroma. Jaffe's 2 patients,[12,13] each with a chondromyxoid fibroma in a rib, were asymptomatic and the diagnosis was incidental to roentgen examination of the chest. The roentgen evidence of punctate calcification present in our patient does not support the diagnosis, even though it might be suggested by the radiolucency and expansion. As Jaffe points out, the distinctions between chondromyxoid fibroma, with or without malignant transformation, and chondrosarcoma of primary type are often difficult to draw. R. O.'s lesion represents one in which histologic diagnosis is difficult, and in which the future course of the patient may illuminate the pathology further.

In our Case 11, there was also some disagreement concerning the exact histologic diagnosis. The authors and most of the

consultants who saw the lesion thought it represented an osteoblastoma. Several others felt that the lesion was not a tumor, but rather an abnormal reparative reaction subsequent to a fracture. In support of this latter possibility is the known occurrence of fractures in irradiated bone. This has been reported not only in the femoral neck after roentgen treatment of carcinoma of the cervix, but also in ribs following therapy for breast carcinoma. The time interval between cessation of treatment and the development of the lesion also corresponds to that described for post-irradiation fractures (usually one to five years), rather than that described for post-irradiation tumors (often more than eight years). However, most of the reported fractures were not examined histologically. Roentgenologically, they did not resemble the lesion under discussion, nor were they preceded by the appearance of an expanding radiolucent focus in the rib. It is probable that the fracture seen in the patient under discussion was secondary to the replacement of bone by nonosseous tissue, probably neoplastic.

Irradiation of growing bones can lead to disturbances in normal skeletal development. Some of these complications, *e.g.*, scoliosis and exostosis, have been reported by Neuhauser *et al.*[25] and are exemplified by our patients. They both have postirradiation scoliosis and underdevelopment of the ilium. In addition, Case II developed an osteochondroma of the right ilium five years after roentgen treatment.

One case reported by Cruz *et al.*[5] is noteworthy in that the patient showed a post-irradiation osteogenic sarcoma in a clavicle and, in addition, an osteochondroma of the adjoining humerus. These complications, together with the relatively high frequency of postirradiation neoplasms, emphasize the need for prolonged and careful surveillance of all children receiving radiation therapy.

SUMMARY

In a selected series of 207 children who received roentgen therapy with 1,000 r or more delivered to bone, 2 subsequently developed neoplasms of bone. Both bone tumors occurred in normal ribs included in the field of treatment. The tumors were atypical; one resembled a chondrosarcoma, the other an osteoblastoma. The latent periods were thirteen and two years, respectively. Both children had postirradiation scoliosis and hypoplasia of the ilium. One also developed an osteochondroma of the ilium.

Jonathan Cohen, M.D.
300 Longwood Avenue
Boston 15, Massachusetts

REFERENCES

1. Beck, A. Zur Frage des Röntgensarkoms, zugleich ein Beitrag zur Pathogenese des Sarkoms. *München. med. Wchnschr.*, 1922, *69*, 623–624.
2. Cade, S. Radiation induced cancer in man. *Brit. J. Radiol.*, 1957, *30*, 393–402.
3. Cahan, W. G., Woodard, H. Q., Higinbotham, N. L., Stewart, F. W., and Coley, B. L. Sarcoma arising in irradiated bone; report of 11 cases. *Cancer*, 1948, *1*, 3–29.
4. Copeland, M. M. Effect of ionising radiations on bone (other than marrow), and on striated muscle. *Am. Acad. Orthop. Surgeons*, Lect., 1953, *10*, 191–212.
5. Cruz, M., Coley, B. L., and Stewart, F. W. Postradiation bone sarcoma. *Cancer*, 1957, *10*, 72–88.
6. Denks, H. Zur Frage des Röntgensarkoms. *Arch. f. klin. Chir.*, 1931, *168*, 215–227.
7. DeYoung, R. Development of sarcoma in bone subjected to irradiation; case report. *Am. Surgeon*, 1952, *18*, 816–819.
8. Francisco, C. B., Pusitz, M. E., and Gerundo, M. Malignant degeneration of benign bone cyst? *Arch. Surg.*, 1936, *32*, 669–678.
9. Glasser, O., Quimby, E. H., Taylor, L. S., and Weatherwax, J. L. Physical Foundations of Radiology. Second edition. Paul B. Hoeber, Inc., New York, 1952, p. 519.
10. Gruca, A. Przypadek miesaka kolana na tle gruzlicy, leczonej promieniami Roentgena. (Case of sarcoma developing after roentgenotherapy of tuberculosis of bones.) *Chir. narz. ruchu*, 1934, *7*, 185–187 (Abst.: *Ztschr. f. Orthop.*, 1936, *65*, 89).
11. Hatcher, C. H. Development of sarcoma in bone subjected to roentgen or radium irradiation. *J. Bone & Joint Surg.*, 1945, *27*, 179–195.
12. Jaffe, H. L. Tumors and Tumorous Conditions of the Bones and Joints. Lea & Febiger, Philadelphia, 1958.

13. JAFFE, H. L. Giant cell tumor of bone; problems of differential diagnosis. *Bull. Hosp. Joint Dis.*, 1944, *5*, 84–91.
14. JARUSLAWSKY, W. Kniegelenkstuberkulose und Sarkom. *Zentralbl. f. Chir.*, 1929, *56*, 915–917.
15. JONES, A. Irradiation sarcoma. *Brit. J. Radiol.*, 1953, *26*, 273–284.
16. KÜTTNER, H. Zur Frage der Geschwulstentstehung nach Röntgenbestrahlung von Gelenk- und Knochentuberkulosen. *Arch. f. klin. Chir.*, 1931, *164*, 5–38.
17. LACASSAGNE, A. Conditions dans lesquelles ont été obtenus, chez le lapin, des cancers par action des rayons X sur des foyers inflammatoires. *Compt. rend. Soc. de biol.*, 1933, *112*, 562–564.
18. LACASSAGNE, A., and VINZENT, R. Sarcomes provoqués chez des lapins par l'irradiation d'abcès a *Streptobacillus caviae*. *Compt. rend. Soc. de biol.*, 1929, *100*, 249–251.
19. LICHTENSTEIN, L. Bone Tumors. Second edition. C. V. Mosby Co., St. Louis, 1959.
20. LICHTENSTEIN, L. Aneurysmal bone cyst; further observations. *Cancer*, 1953, *6*, 1228–1237.
21. LÜDIN, M. Knochensarkom nach experimenteller Röntgenbestrahlung. *Acta radiol.*, 1934, *15*, 553–556.
22. MARIE, P., CLUNET, J., and RAULOT-LAPOINTE, G. Contribution à l'étude du développement des tumeurs malignes sur les ulcères de Roentgen. *Bull. Assoc. franç. p. l'étude du cancer*, 1910, *3*, 404–426.
23. MARSCH, E. Tuberkulose und Sarkom (Röntgensarkom?). *Zentralbl. f. Chir.*, 1922, *49*, 1057–1060.
24. National Bureau of Standards Handbook 62. Report of the International Commission on Radiological Units and Measurements (ICRU) 1956, p. 17.
25. NEUHAUSER, E. B. D., WITTENBORG, M. H., BERMAN, C. Z., and COHEN, J. Irradiation effects of roentgen therapy on growing spine. *Radiology*, 1952, *59*, 637–650.
26. SABANAS, A. O., DAHLIN, D. C., CHILDS, D. S., JR., and IVINS, J. C. Postradiation sarcoma of bone. *Cancer*, 1956, *9*, 528–542.
27. SKOLNIK, E. M., FORNATTO, E. J., and HEYDEMANN, J. Osteogenic sarcoma of skull following irradiation. *Ann. Otol. Rhin. & Laryng.*, 1956, *65*, 915–936.
28. TEBBETT, R. D., and VICKERY, R. D. Osteogenic sarcoma following irradiation for retinoblastoma with report of case. *Am. J. Ophth.*, 1952, *35*, 811–818.

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CONGENITAL BIFID STERNUM WITH PARTIAL ECTOPIA CORDIS*

By C. H. (JOSEPH) CHANG, M.D., *and* W. CLAYTON DAVIS, M.D.
MAN, WEST VIRGINIA

PARTIAL ectopia cordis with congenital bifid sternum is, according to the current literature, a difficult problem for surgical repair and reconstruction. Sabiston,[8] in 1958, reported his successful repair in a child two years of age. The purpose of this paper is to present the case report of a child operated upon in the fourth year of life, using the method of Sabiston, and to discuss the radiologic findings associated with this defect.

REPORT OF A CASE

W.V.F., a four year old white boy, had been noted to have absence of the sternum in the midline since birth. The mother had reported pulsations beneath the skin and the condition had been undiagnosed until a local physician referred him for thoracic surgical evaluation. The child was the thirteenth of 14 pregnancies and there was no family history of any other known congenital deformities. The past medical history was entirely negative except for short episodes of upper respiratory infections and tonsillitis. The physical findings were normal except for the anomaly of the chest. This showed a prominent V-shaped defect in the area of the sternum. The two medial clavicular heads were separated by 5.5 cm. when the patient was in a normal standing position. The xiphoid process was present and united. The edges of the deformity were composed of two sternal bodies. Coughing produced marked bulging in the area of the defect, showing evidence of bilateral pulmonary herniation into the mediastinum. Pulsation of the heart and great vessels was easily detectable by both inspection and palpation. No cardiac abnormalities were discovered. Examination of the skin over the defect showed a 1.5 cm. circular area suggestive of a scar. The skin in this area seemed to be fibrous in quality. There was no drainage present and no past history of any drainage. This area had been visible since birth according to the mother. Complete blood cell count was normal except for eosinophilia. Blood chemistries were within normal limits. Urinalysis was normal. Serology was nonreactive. Stools for ova and parasites showed a few ova of Trichuris trichiura. Electrocardiogram was normal. Chest roentgenograms (Fig. 1, *A* and *B*) showed a wide separation of the sternal ends of both clavicles and widening of the superior mediastinal shadow in the posteroanterior view. This finding was compatible with a clinical diagnosis of bifid sternum with partial ectopia cordis. In the lateral view (Fig. 2, *A* and *B*) there was absence of the manubrial shadow and loss of convexity of the manubrio-sternal junction with concavity in the suprasternal region. Esophagram was within normal limits.

On August 20, 1959 repair of the partial ectopia cordis and bifid sternum was performed under general endotracheal anesthesia. A midline incision was made, extending from 3 cm. below the thyroid cartilage down to the tip of the joined xiphoid process. Full flaps of skin and subcutaneous tissue were raised and the pericardium was visualized beneath these flaps. Skin flaps, subcutaneous tissue and then the muscle layers were reflected from the ribs. The pericardium was freed from the under surface of the unjoined sternal bodies. The perichondrium was freed anteriorly and posteriorly from the cartilages. A triangular wedge of the connected xiphoid was excised. Two temporary sutures were placed around the sternal bodies, and, when these were tied, they reduced the defect approximately one-half its greatest width. The first four costal cartilages on either side were then freed and exposed subperichondrially. Oblique incisions were made in each of these cartilages and circumferential sutures around the bifid sternal bodies completely approximated the defect due to the relaxation afforded by the oblique division of the four superior costal cartilages. The posterior and anterior portions of sternal periosteum were approximated. Circumferential sutures around the sternal bodies were tightly joined. There was no evidence of entrance into the pleural cavities.

* From the Radiological and Surgical Services, Man Memorial Hospital, Man, West Virginia.
† Present address: Assistant Professor of Surgery, University of Pittsburgh School of Medicine, and Assistant Chief, Surgical Service, V. A. Hospital, Pittsburgh, Pennsylvania.

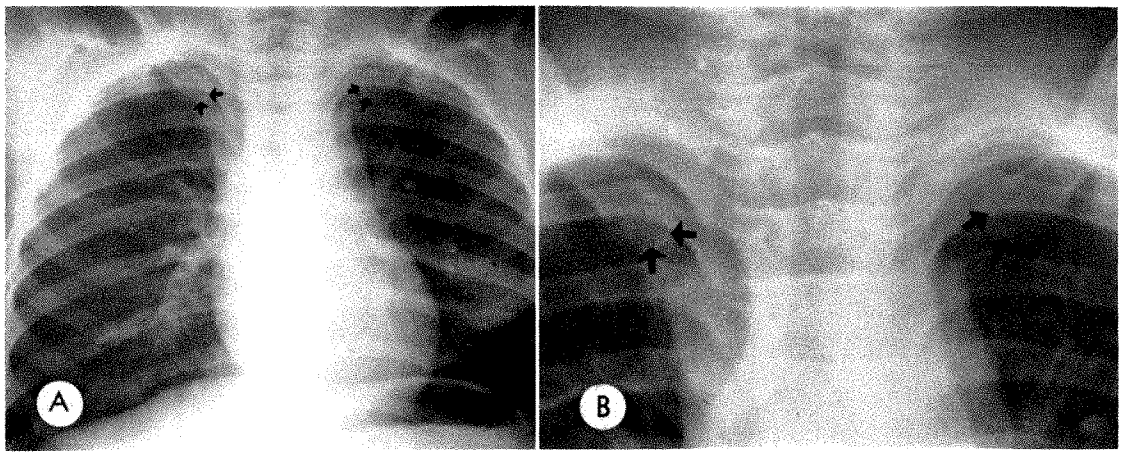


FIG. 1. (*A*) Preoperative roentgenogram of the chest showing wide separation of the sternal ends of the clavicles (arrows) and widening of the superior mediastinal shadow. (*B*) Enlargement of the area of abnormality shown in *A*.

The subcutaneous tissue and skin were approximated.

Motion pictures were made of the entire operative procedure and the immediate postoperative result showed a good functional and cosmetic repair of the preoperative defect. On the evening of operation, a 50 per cent right pneumothorax appeared, and this was completely relieved by aspiration of air from the right pleural cavity. On the first postoperative day, a 30 per cent left pneumothorax was immediately relieved by thoracentesis. Following this, both lung fields remained clear and normal. The boy was given intensive preoperative and postoperative physical therapy and was discharged from the hospital on the seventh postoperative day, completely asymptomatic. One year following operation, physical examination showed an adequate cosmetic and functional recovery. Follow-up roentgenograms (Fig. 3, *A*

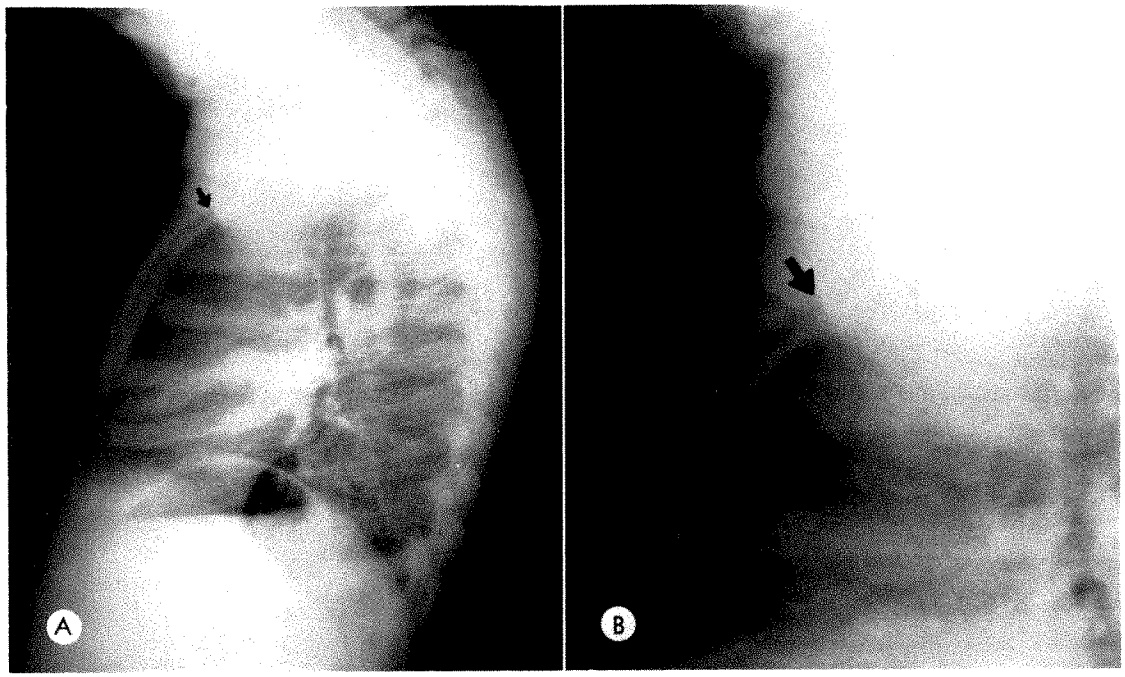


FIG. 2. (*A*) Preoperative left lateral roentgenogram of the chest showing absence of the manubrial shadow and loss of convexity of the manubrio-sternal junction. There is a concavity in the suprasternal region (arrow). (*B*) Enlargement of the area of abnormality shown in *A*.

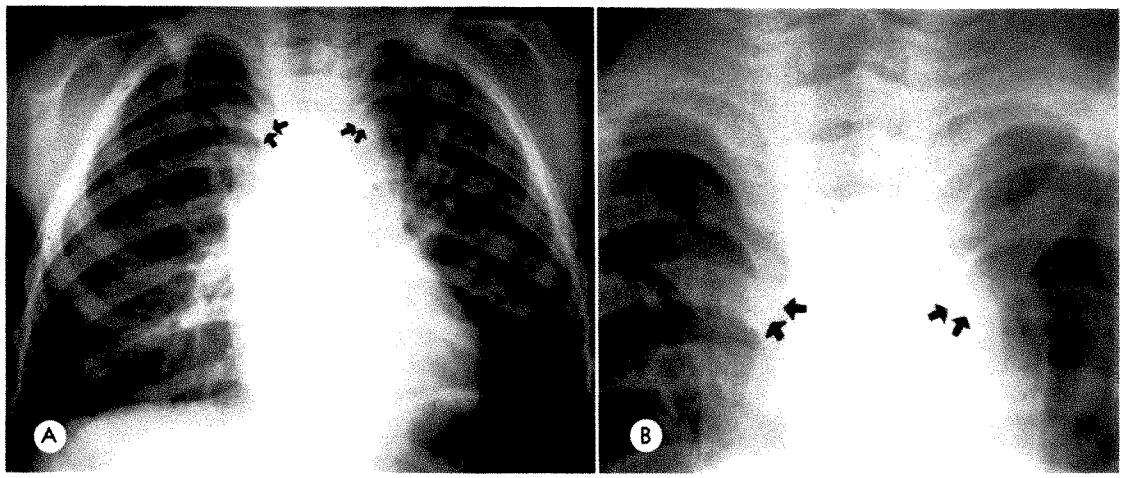


Fig. 3. (*A*) Postoperative posteroanterior roentgenogram of the chest showing approximation of the sternal ends of the clavicles (arrows). (*B*) Enlargement of area shown in *A*.

and *B*, and 4) also showed approximation of the sternal ends of the clavicles.

DISCUSSION

EMBRYOLOGY

In 1885 Madden,[6] reporting a case of congenital fissure of the sternum, was the first to state that the condition was due to a failure of union of the two primitive cartilages from which the osseous sternum is formed. He remarked that this same imperfection was quite frequently seen in hare-lip, often extending to the osseous structures beneath, such as the superior maxillary bone. Spina bifida and exstrophy of the bladder were felt by him to be other examples of the same type of failure of fetal development and nonunion in the midline. Hanson,[3] in discussing the ontogeny and phylogeny of the sternum in animals and man, added much to our understanding of congenital bifid sternum. He demonstrated that the sternum originates separately from the ribs as two longitudinal structures in the anterior thorax. The space between these two sternal bodies is present from the sixth to the ninth week embryo. The bands also fuse laterally with the costal cartilages. It is therefore apparent that in the rare instances of congenital bifid sternum there is a failure of this midline union. A defect results and through it the pericardium may herniate, being covered only by subcutaneous tissue and skin. This is the condition demonstrated in our present case. Gussow,[2] in 1946, first described the surgically unexplained scar-like depression in the midportion of the anterior chest wall. Since then this has been noted by other authors and was definitely present in our case. He remarked that this round scar-like depression suggests an anomaly of the sternum.

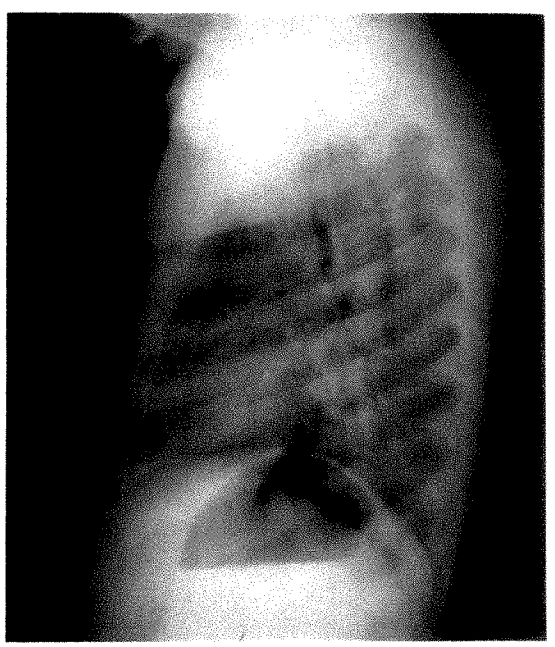


Fig. 4. Postoperative left lateral roentgenogram of chest.

ROENTGENOGRAPHIC FINDINGS

Though Gussow[2] stated that the routine roentgenograms of the chest are not of much help in this anomaly, the frontal view of the chest is definitely of diagnostic value.

Wide separation of the sternal ends of the clavicles and widening of the superior mediastinal shadow on the posteroanterior or anteroposterior roentgenograms of the chest are characteristic features of this anomaly (Fig. 1, *A* and *B*). The manubrial shadow is absent. On the lateral roentgenogram there is seen a loss of the usual convexity of the manubrio-sternal junction and the presence of a concavity in the suprasternal region (Fig. 2,*A* and *B*).

SURGICAL CONSIDERATIONS

In 1947, Burton[1] reported his experience in the surgical treatment of congenital bifid sternum with partial ectopia cordis. Both of his cases were treated by a block of cartilage which was removed from the costal margin and placed over the defect. The cosmetic results were only moderately good. In 1949, Maier and Bortone[7] performed a primary surgical closure. A good result was obtained by a direct suture procedure in a six week old infant. In a discussion of Maier and Bortone's paper, Klassen[4] reported a similar case where an attempt at repair was unsuccessful. Longino and Jewett[5] described a successful primary approximation of a bifid sternum in a six day old infant. It is noted that there is general agreement that whenever possible the preferred management should be direct operative closure in early infancy. Sabiston[8] first reported a logical method of repair of congenital bifid sternum with ectopia cordis in older children. The repair which he described was the one used in our case and to our knowledge from a review of the literature the only other case thus treated. Our cosmetic and functional results were considered successful; this was also true in the case reported by Sabiston. The operation was considered simple and effective. The sliding costal cartilage technique offered no difficulty in the correction of the deformity. The present authors agree that with Sabiston's technique there is no necessity for the use of tissue grafts or plastic materials in the repair of this malformation.

SUMMARY

1. A case of successful repair of congenital bifid sternum with partial ectopia cordis in a four year old child has been presented. This is the second successful case known to have been repaired by this method.

2. Bifid sternum with partial ectopia cordis is a very rare anomaly, resulting from failure of union of the two primitive sternal bands.

3. The characteristic roentgen findings are wide separation of the sternal ends of the clavicles and widening of the superior mediastinal shadow in the frontal view of the chest and loss of normal convexity at the manubrio-sternal junction in the lateral view. The manubrial shadow is absent.

C. H. (Joseph) Chang, M.D.
Man Memorial Hospital
Man, West Virginia

REFERENCES

1. BURTON, J. F. Method of correction of ectopia cordis. *Arch. Surg.*, 1947, *54*, 79–84.
2. GUSSOW, L. Congenital anomaly of sternum—vertical non-fusion; report of two cases. *Military Surg.*, 1946, *98*, 420–424.
3. HANSON, F. B. Ontogeny and phylogeny of sternum. *Am. J. Anatomy*, 1919, *26*, 41–115.
4. KLASSEN, K. Discussion of Maier and Bortone.[7]
5. LONGINO, L. A., and JEWETT, T. C., JR. Congenital bifid sternum. *Surgery*, 1955, *38*, 610–614.
6. MADDEN, F. Congenital fissure of sternum. *New York Med. J.*, 1885, *41*, 406–409.
7. MAIER, H. C., and BORTONE, F. Complete failure of sternal fusion with herniation of pericardium; report of case corrected surgically in infancy. *J. Thoracic Surg.*, 1949, *18*, 851–859.
8. SABISTON, D. C., JR. Surgical management of congenital bifid sternum with partial ectopia cordis. *J. Thoracic Surg.*, 1958, *35*, 118–122.

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CONGENITAL PARTIAL DEFECT OF THE PERICARDIUM ASSOCIATED WITH HERNIATION OF THE LEFT ATRIAL APPENDAGE*

By C. H. (JOSEPH) CHANG, M.D.,† *and* TED F. LEIGH, M.D.
ATLANTA, GEORGIA

CONGENITAL pericardial defect is a very rare anomaly, although Columbus[7] described a case of pericardial defect as early as 1559. The first verified case of congenital absence of the pericardium was reported by Baillie[1] in 1793.

In 1925, Moore and Shumacker[17] reported 64 recorded cases of periocardial defect. More recently Ellis, Leeds, and Himmelstein[9] compiled a total of 99 cases from the medical literature including 2 of their own. The present case report, together with a recent report of 5 such cases by Jones,[12] a single case by Brea *et al.*,[4] and 2 cases by Hering *et al.*,[11] brings the total number to 108.

Partial pericardial defect is less common than complete absence of the pericardium. In some instances when there is a partial defect, there may also be herniation of all or a portion of the heart, and this may lead to the patient's death.

Syncope and sudden death were reported in one case by Boxall[3] and in another by Sunderland and Wright-Smith;[27] in these cases, incarceration and strangulation of the heart were caused by a hernial ring of the pericardium. In our present case, there was a herniation of the left atrial appendage through a partial pericardial defect. Similar cases have been reported, one by Keith,[13] one by Osgood and Spector,[18] and recently 2 by Hering *et al.*[11]

The purpose of this paper is to describe certain characteristic roentgenologic features of congenital pericardial deficiency with herniation of the left atrial appendage and aid in the differential diagnosis of this abnormal configuration of the cardiac silhouette.

REPORT OF A CASE

R.E.H. was a six year old white male who was admitted to Emory University Hospital for evaluation of a cardiac murmur which had been heard by the patient's pediatrician six months earlier. The patient had a history of easy fatiguability and occasional paleness. Physical examination revealed a Grade IV murmur in the pulmonary area which was continuous through systole and diastole. There was radiation of this murmur into both sides of the neck, the left shoulder and the back. There was no cyanosis or clubbing, and the patient was normally developed and nourished. The family history was noncontributory.

Cardiac fluoroscopy and chest roentgenograms disclosed a heart of normal size. The pulmonary blood flow was slightly increased and the aortic knob was prominent—two findings which suggested the diagnosis of patent ductus arteriosus (Fig. 1, *A* and *B*). In addition to these findings, there was also noted a sharply defined prominence on the left heart border just caudad to the main pulmonary artery segment, which showed active pulsations paradoxical to those in the aorta and pulmonary artery.

At operation, a patent ductus arteriosus was identified, and this was incised and ligated. In addition, the prominence on the left cardiac border was found to be a herniation of the left atrial appendage through a pericardial foramen (Fig. 2). This defect was covered with a flap of mediastinal pleura.

Postoperatively, the patient developed pericarditis with effusion on the seventh day. This was treated with antibiotics, and repeated pericardicentesis. On the fourteenth postoperative day, he was discharged in good condition, and has remained well since.

INCIDENCE

Partial pericardial anomalous defects

* From the Department of Radiology, Emory University School of Medicine, Atlanta, Georgia.
† Present address: Man Memorial Hospital, Man, West Virginia.

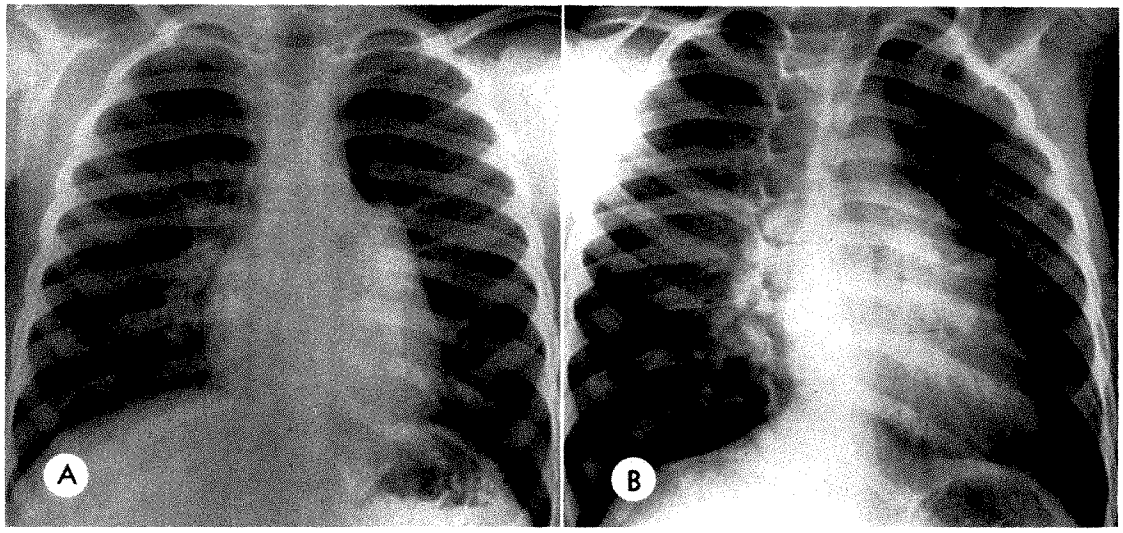


FIG. 1. (*A*) Posteroanterior chest roentgenogram shows a sharply defined and smooth prominence on the left cardiac border just caudad to the main pulmonary artery segment; active pulsations paradoxical to those in the aorta and pulmonary artery were seen during cardiac fluoroscopy. The pulmonary flow appears slightly increased and the aortic knob is somewhat prominent. (*B*) Right anterior oblique roentgenogram of the chest also shows the smooth prominence just caudad to the main pulmonary artery segment.

have been encountered most frequently on the left side. In 108 cases recorded in the literature, the anomaly was left sided in 73 (67 per cent) and bilateral in 3.

Complete or almost complete defects with common pericardial and left pleural cavities are more commonly seen. In 46 cases reported by Southworth and Stevenson,[26] 34 (76 per cent) had a complete defect in the pericardium, and in the remaining cases there was partial absence of this structure, which gave the appearance of a foramen of varying size.

The anomaly appears to be more frequent in males than in females, the ratio being approximately 3:1.

PATHOGENESIS

Though the embryogenesis of pericardial defects in man is not settled, most authors are in agreement that they represent a failure of closure of the pleural pericardial foramen.

Keith,[13] and Rusby and Sellors[23] have suggested that herniation of an abnormal lung bud through the foramen might prevent its closure. Risel[21] differed with these investigators and suggested as a cause an atresia of the pleural pericardial membrane with failure to join the lung roots. McGarry[16] believed that abnormal tension created by the rotation of the developing liver traumatized the pulmonary ridge from which the membrane develops.

Perna[19] and Plaut[20] proposed a vascular atrophy theory based on the hypothesis that premature atrophy of the left duct of Cuvier is causative in producing a deficiency of the blood supply to the pleural pericardial membrane. The latter theory has been generally accepted because it accounts best for the preponderance of pericardial defects on the left side. The possibility that trauma may also play a part in the production of these defects is raised by Moore and Shumacker's[17] recent case, in which there was a past history of a crushing injury to the chest in an automobile accident when the patient was five years of age.

ROENTGEN FINDINGS

The first roentgen diagnosis of pericardial defect was made by Ellis, Leeds, and Himmelstein[9] in 1958. Dahl[8] in 1937 inferred the presence of a communication between the pericardium and the left

pleural space. Following an artificial pneumothorax for treatment of pulmonary tuberculosis in a man twenty years of age, a roentgenogram revealed a pneumopericardium in addition to a left pneumothorax. Kerley[25] mentioned a case of congenital pericardial defect in which the diagnosis was implied after noting pneumopericardium following a diagnostic pneumothorax on the right for investigation of a congenital lung cyst. Jones[12] also observed accidentally a well marked pneumopericardium following a right sided diagnostic pneumothorax for a mediastinal mass.

There are several different types of roentgenologic examinations which can be performed in the investigation of pericardial defects. These will be discussed briefly.

1. *Routine Chest Examination.* In partial defects of the pericardium, the roentgen findings are variable, depending on whether or not there is associated herniation of the heart or lung. If there is a herniation of the heart or lung, the herniated part appears as a mass projecting from the cardiac border. Since the pericardial sac, in the frontal projection, extends between the level of the third through the seventh ribs in their anterior aspect, the mass should be located within this level. The size and the shape of the herniated mass is dependent on the size of the foramen and the herniated part.

In our case, the herniation is best shown in a slight right anterior-oblique view of the chest (Fig. 1*B*). The roentgen findings in the cases reported by Hering *et al.*[11] are identical to those of our case. In the case reported by Rusby and Sellors[23] the roentgen findings are also similar but, in theirs, the mass was due to a lung cyst residing in the pericardial defect. Brea *et al.*[4] observed a smooth projection on the left just below the aortic arch. Case 9 of Jones[12] had a right mediastinal mass due to the presence of a lung cyst associated with a pericardial defect.

In those instances where there is a partial pericardial defect without an associated herniation, there is no demonstrable abnormality in the cardiac silhouette. The cases reported by Dahl[8] and by Venturini and Pesci[28] were of this type.



Fig. 2. Photograph taken at operation indicates the rim of the partial pericardial defect (solid arrows) and the herniated left atrial appendage (broken arrows) through the pericardial defect.

In complete pericardial defect the roentgenographic characteristics are quite different and these are well described by Ellis, Leeds and Himmelstein.[9]

2. *Diagnostic Pneumothorax.* This is a most important diagnostic method of investigation for a congenital pericardial defect; the demonstration of a pneumopericardium following induction of an artificial pneumothorax is diagnostic. Several authors (Dahl,[8] Rusby and Sellors,[23] Kerley,[25] and Jones[12]) have reported the accidental demonstration of communications between the pericardial and the pleural space following the introduction of air into the pleural space.

Ellis *et al.*[9] have recommended, for left pericardial defects, a right posterior ob-

lique roentgenogram of the chest taken with the horizontal beam and the left side down, following introduction of 500 cc. of air into the left pleural space. The absence of a pneumopericardium following pneumothorax does not necessarily rule out the presence of a pericardial defect, since in some cases adhesions might prevent the accumulation of air in the pericardial sac.

3. *Cardiac Fluoroscopy.* Fluoroscopy, especially with an image amplifier, may be helpful to differentiate from a mediastinal tumor; and observation of a paradoxical pulsation to those in the aorta and pulmonary artery might aid in establishing a diagnosis. Our case showed marked pulsation of the mass, and it appeared to be apparently paradoxical to the contraction of the pulmonary artery and the aorta. Rusby and Sellors'[23] case also showed the mass to be pulsatile; however, it was distinct from the aorta.

4. *Angiocardiography.* This procedure may help to rule out other cardiac abnormalities and will give positive information concerning the anatomic location of the herniated part in the case of a partial pericardial defect. It will also permit a differential diagnosis between herniated heart and lung. Kjellberg, Mannheimer, Rudhe and Jonsson,[14] in their textbook on congenital heart disease, reproduced an angiocardiogram showing herniation of the right ventricle through a large left pericardial defect in a six year old girl who also had a patent ductus arteriosus. Recently, Hering *et al.*[11] diagnosed a congenital pericardial defect with herniation of the left atrial appendage preoperatively by angiocardiographic study in a twenty-two year old sergeant of the United States Marine Corps.

DISCUSSION

A review of the literature indicates that in the human subject absence of the pericardium is without significance. Moore and Shumacker[17] carried out total or partial resection of the pericardium in 65 dogs and concluded that in none of these animals was there any indication of impaired cardiac function or dilatation of the heart resulting from the procedure.

The main hazard associated with congenital partial pericardial defect appears to be herniation and incarceration of the heart through the pericardial foramen. In 2 reported cases,[3,27] syncope and death within hours occurred from incarceration and strangulation of the heart by the hernial ring of the pericardium.

A complete absence of the pericardium causes no serious disturbance, and may produce variable cardiac murmurs and mild chronic chest symptoms. The two cases of Ellis, Leeds, and Himmelstein[9] had unexplained chest pain, possibly related to the pericardial defect. Superimposed pulmonary or pleural infection, however, will be dangerous.

Other associated congenital anomalies are also reported in the literature. Kjellberg, Mannheimer, Rudhe and Jonsson,[14] Osgood and Spector,[18] and our present case had patent ductus arteriosus. The case of Rusby and Sellors[23] and 5 cases of Jones[12] had bronchogenic cysts. Brea, Croxatto and De All[4] found an aberrant pulmonary lobe associated with pericardial defect. Moore and Shumacker's[17] case had tricuspid insufficiency of unknown etiology. Ladd's[15] case showed an associated large left diaphragmatic hernia.

Patching of the partial pericardial defect with a flap of mediastinal pleura may prevent herniation of the heart and incarceration by the herniated ring of the pericardium. The partial defect in our case was covered with a flap of mediastinal pleura. Though, postoperatively, this patient developed pericarditis with effusion, he was treated satisfactorily with antibiotics and pericardicentesis. Wide resection of that portion of the pericardium could also prevent herniation and may eliminate immediate postoperative complications.

In addition to the congenital pericardial defect, the acquired herniation as a sequela to intrapericardial surgery, is frequently encountered.

SUMMARY

A case of congenital partial pericardial defect associated with herniation of the left atrial appendage is presented. This patient also had a patent ductus arteriosus.

The literature pertaining to this abnormality is reviewed, and incidence, pathogenesis and roentgen findings are discussed.

Congenital absence of the pericardium is a rare anomaly. In the majority of reported cases, it was found on the left side (67 per cent). Three cases having defects on both sides are reported in the literature. The anomaly is more frequent in males than in females, the ratio being approximately 3:1.

The roentgen findings in this anomaly show certain characteristics. On the routine chest roentgenogram, an abnormal cardiac configuration is seen, there being a smooth projection from the left cardiac border just caudad to the main pulmonary artery segment. On fluoroscopic examination pulsations are noted in the mass, which appear to be paradoxical to those of the pulmonary artery and aorta. The demonstration of pneumopericardium following induction of artificial pneumothorax is diagnostic. Angiocardiographic studies are helpful in determining the anatomic site of the herniation through the pericardial defect.

Repair of the pericardial foramen with mediastinal pleura may prevent herniation and incarceration of the heart. It is possible, however, that wide resection of that portion of the pericardium may also prevent incarceration and strangulation of the heart.

C. H. (Joseph) Chang, M.D.
Man Memorial Hospital
Man, West Virginia

REFERENCES

1. Baillie, N. On want of pericardium in human body. *Tr. Soc. Imp. Med. & Chir. Knowledge*, 1793, *1*, 91–102.
2. Beck, C. S. Congenital deficiency of pericardium. *Arch. Surg.*, 1931, *22*, 282–288.
3. Boxall, R. Incomplete pericardial sac; escape of heart into left pleural cavity. *Tr. Obst. Soc.*, 1887, *28*, 209–210.
4. Brea, M. M., Croxatto, O. C., and De All, J. A. Lóbulo pulmonar aberrante con defecto pericardico. *Dia med.*, 1958, *30*, 2734–2736.
5. Burke, D. M., and Bradshaw, D. E. Congenital anomaly of pericardium due to lack of separation of right pericardial wall from chest wall. *Bull. Internat. A. M. Mus.*, 1950, *31*, 178–185.
6. Chiodin, L. A. Asplasis congénita de la hoja parietal del pericardio. *Rev. Soc. argent. biol.*, 1932, *8*, 361–367.
7. Columbus, M. R. De re anatomica. Vol. XV. 1559, p. 265.
8. Dahl, E. Et. tilfelle av medfodt pericarddefekt diagnostisert after anleggav venstresidig pneumothorax. *Med. rev., Bergen*, 1937, *54*, 312–315.
9. Ellis, K., Leeds, N. E., and Himmelstein, A. Congenital deficiencies in parietal pericardium; review with 2 new cases including successful diagnosis by plain roentgenography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 125–137.
10. Gruber, C. B. Zur Kenntnis kranialer Doppelbildung beim Menschen mit Zwerchfellsfehlern und mit Herzbeutel-Lunge. *Beitr. path. Anat.*, 1949, *110*, 346–360.
11. Hering, A. C., Wilson, J. S., and Ball, R. E., Jr. Congenital deficiency of pericardium. *J. Thoracic Surg.*, 1960, *40*, 49–55.
12. Jones, P. H. Developmental defects in lungs. *Thorax*, 1955, *10*, 205–213.
13. Keith, J. A. Partial deficiency of pericardium. *J. Anat. & Phys.*, 1907, *41*, 6–7.
14. Kjellberg, S. R., Mannheimer, E., Rudhe, U., and Jonsson, B. Diagnosis of Congenital Heart Disease. Year Book Publishers, Inc., Chicago, 1955.
15. Ladd, W. E. Congenital absence of pericardium. *New England J. Med.*, 1936, *214*, 183–187.
16. McGarry, K. A. Case of patency of pericardium and its embryological significance. *Anat. Rec.*, 1914, *8*, 43–54.
17. Moore, T. C., and Shumacker, H. B., Jr. Congenital and experimentally produced pericardial defects. *Angiology*, 1953, *4*, 1–11.
18. Osgood, R., and Spector, B. Defective pericardial sac and interatrial septum and atresia of pulmonic orifice. *Am. J. Dis. Child.*, 1941, *61*, 1028–1033.
19. Perna, G. Sopra un arresto di sviluppo della sierosa pericardica nell'uomo. *Anat. Anz.*, 1909–1910, *35*, 323–338.
20. Plaut, M. Ueber zwei weitere Fälle von Defekt des Herzbeutels. *Frankfurt. Ztschr., f. Path.*, 1913, *12*, 141–156.
21. Risel. Canalis neurentericus und Rhachischisis anterior (Drei Fälle von Persistenz des Canalis neurentericus bei Rhachischisis anterior mit

Störungen in Schluss des Zwerchfells). *Verhandl. d. deutsch. path. Gesellsch.*, 1912, *15*, 379–415.

22. RONKA, E. K. F., and TESSMER, C. F. Congenital absence of pericardium. *Am. J. Path.*, 1944, *20*, 137–141.
23. RUSBY, N. L., and SELLORS, T. H. Congenital deficiency of pericardium associated with bronchogenic cyst. *Brit. J. Surg.*, 1945, *32*, 357–364.
24. SHAFIROFF, B. G. B. Congenital absence of pericardium; case report. *J. Thoracic Surg.*, 1951, *21*, 30–33.
25. SHANKS, S. C., and KERLEY, P. Textbook of X-Ray Diagnosis. Vol. II. Second edition. W. B. Saunders Company, Philadelphia, 1951, pp. 146–147.
26. SOUTHWORTH, H., and STEVENSON, C. S. Congenital defects of pericardium. *Arch. Int. Med.*, 1938, *61*, 223–240.
27. SUNDERLAND, S., and WRIGHT-SMITH, R. J. Congenital pericardial defects. *Brit. Heart J.*, 1944, *6*, 167–175.
28. VENTURINI, A., and PESCI, A. Le agenesie del pericardio. *Arch. chir. torace*, 1957, *14*, 189–202.

VOL. 86, NO. 3

ROENTGENOGRAPHIC FEATURES OF ASPLENIA, A TERATOLOGIC SYNDROME OF VISCERAL SYMMETRY*

By WILLIAM J. FORDE, M.B., *and* NATHANIEL FINBY, M.D.†
NEW YORK, NEW YORK

CONGENITAL absence of the spleen is often part of a syndrome of congenital anomalies consisting of malformations of the heart, partial situs inversus, symmetric lobulation of the liver and the lungs, anomalies of arteries and veins and persistence of the dorsal mesentery to the duodenum and colon.[10,13,14,15] This is a rare syndrome, but has been reported in the medical literature with increasing frequency in recent years.[1,9,14]

Putschar and Manion[15] reviewed the literature in 1956 and noted that 78 cases of congenital absence of the spleen had been reported; of these 29 cases were associated with malformations of the heart and partial situs inversus, and 20 cases were associated with cardiac malformations without situs inversus. Polhemus and Schafer[14] in 1959 were the first to point out that such congenital anomalies and absence of the spleen have a relationship. An excellent monograph was written by Ivemark[10] in 1955 in which he collected the cases in the literature from the first case of Martin (1826) and added 14 cases of his own. Ivemark[10] noted that congenital asplenia was found only 12 times in 7,032 necropsies at the Boston Children's Hospital; associated anomalies of the heart and great vessels were present in 11 of these instances. He suggested that the syndrome of asplenia associated with malformations should be termed "asplenia, a teratologic syndrome of visceral symmetry." He postulated that the basic lesion of the heart occurred during organogenesis when the spleen should have been in its early formative stage of development.

EMBRYOLOGIC CONSIDERATIONS

To better understand this syndrome, it is pertinent to briefly review the embryology of the spleen. By the 14 mm. (six weeks) stage the spleen can be identified as a projection from the mesogastrium between the pancreas and stomach.[6,17] The splenic anlage first appears as a mesodermal condensation in the dorsal meso gastrium. From this mesogastrium several splenic anlagen differentiate. The fusion of these splenic aggregates usually forms a single normal spleen.

Embryologically, the association of the anomalies can be related to the developmental arrest and malformation initiated during the fifth to seventh week of gestation.[10,14] At this time the splenic primordia develop as noted, the heart separates incompletely into two chambers, the definitive pulmonary lobes are forming and the intestine, previously freely movable within the scope of its restraining mesentery, begins its definitive rotation and attachment.[2,14] Ivemark[10] postulates that the adverse causes which may be responsible for dystrophy of the splenic primordium likewise affect the heart and other organs at this early stage. A tendency towards visceral symmetry as noted in this symdrome and the absence of the spleen, the only unilateral mesenchymal organ of the body, was emphasized by Putschar and Manion,[15] who consider the disturbance to be fundamentally a deviation from the normal development of right and left alaterality, resulting usually in various degrees of bilateral right sidedness. The frequent finding of persistent truncus arteriosus, trans-

* From the Department of Radiology, The New York Hospital—Cornell Medical Center, New York, New York.
† Director of Radiology, St. Luke's Hospital, New York, New York.

position of the great arteries, pulmonary atresia or stenosis, various anomalies of the atrioventricular canal, especially complete common atrioventricular canal and cor biloculare, has led to the consideration that the cardiac manifestations represent abnormal septation of the conotruncus and abnormal division of the atrioventricular canal.

Approximately 100 cases of this syndrome have now been described.[14] Congenital absence of the spleen is a rare anomaly which, in adults, is usually discovered unexpectedly at operation or necropsy. Most cases are seen in young subjects because associated developmental abnormalities are apt to shorten life. The most frequent of these are cardiac malformations and partial situs inversus of the abdominal organs. These were present separately or together in 49 of the 79 cases reviewed by Putschar and Manion.[15] Because of the serious and complex nature of the cardiac malformations associated with asplenia, only 4 of these 79 patients lived three or more years. The majority of the patients were males. In the vast majority of reported cases the diagnosis was made at autopsy.

MATERIAL

Five patients with complete absence of the spleen have been autopsied at The New York Hospital. These are tabulated in Table I and reported below.

The cardiovascular findings are of particular interest. All 5 patients had cyanotic heart disease due to cor biloculare. Associated cardiovascular abnormalities included a right sided aortic arch, a bilateral superior vena cava, partial situs inversus, pulmonary stenosis and atresia and anomaly of brachiocephalic vessels.

One patient had symmetric lobation of the liver and another a centrally placed liver. Three patients showed abnormal lobation of the lungs.

Partial situs inversus was present in 3 of the 5 patients, confirming the statements [4,8,14] that a partial situs inversus of the abdominal viscera in a patient with cyanotic heart disease and the hematologic abnormalities of asplenia indicate that the

TABLE I

FINDINGS IN 5 CASES OF ASPLENIA AUTOPSIED AT THE NEW YORK HOSPITAL

Case No.	Clinical Data	Cardiovascular Findings	Other Autopsy Findings
I.	White male, aged 4 yr. Cyanosis, congestive failure and repeated infections	Cor biloculare; anomalous origin of brachiocephalic vessels	Malrotation of colon; abnormal lung lobation; absent spleen
II.	White female, aged 3 mo. Cyanosis and congestive failure	Cor biloculare; pulmonary stenosis; bilateral superior vena cava; right aortic arch	Dextroposition of stomach; malrotation of cecum; symmetric lobation of lungs; absent spleen
III.	White female, aged 21 yr. Cyanotic since birth, repeated infections and congestive heart failure	Cor biloculare; right aortic arch; bilateral superior vena cava	Centrally placed liver; partial situs inversus; right sided stomach; absent spleen
IV.	Negro female, aged 2 mo. Cyanosis and dyspnea	Cor biloculare; right aortic arch; dextrocardia	Incomplete situs inversus; bilaterally symmetric liver; right sided stomach; absent spleen
V.	White male, aged 2 da. Cyanosis	Cor biloculare; atresia of pulmonary ostium; patent ductus arteriosus	Symmetric lobation of lungs; absent spleen

heart lesion is of the cor biloculare type. Angiocardiographic studies were performed in 2 patients and showed abnormality compatible with situs inversus plus dextroposition of the aorta in Case I and a left superior vena cava in Case III.

It is of interest to note that there were also 2 cases of hyposplenia in the autopsy files of The New York Hospital. One was associated with anomalies in the cardiovascular system; namely, defects in the interatrial and interventricular septa, right sided aortic arch and anomalous pulmonary veins. The other patient had a coarctation of the aorta of the infantile type and a patent foramen ovale. In addition, Brandt and Liebow[2] noted that similar but less complex anomalies may be found in polysplenia.

REPORT OF CASES

CASE I. This male infant was seen at The New York Hospital on March 20, 1955 at the age of three months because of dyspnea and cyanosis. He was the product of a full term normal delivery and the mother's prenatal course was uneventful. He had been essentially well until the onset of an upper respiratory infection two days before admission.

He was noted to be acutely ill and had rapid, irregular respirations. The heart was enlarged almost to the anterior axillary line, a precordial bulge was noted and a thrill was felt to the left of the sternum. There was a systolic murmur, maximum in the third left interspace. He was digitalized and given antibiotics and oxygen. Angiocardiography was performed on April 12, 1955 and showed a single large atrium and a large ventricle with dextroposition of the aorta (Fig. 1). He showed satisfactory response to therapy and was discharged on April 29, 1955.

After discharge the patient had repeated bouts of upper respiratory infection and gained weight slowly. Development was somewhat retarded.

Laboratory studies on January 24, 1956 showed the blood hemoglobin to be 10.5 gm. per cent; white blood cell count 28,000 with 68 per cent lymphocytes, 1 per cent monocytes, 2 per cent basophils, and 29 per cent polymorphonuclear leukocytes. Blood smear showed hypochromia, target cells, polychromatic cells and spherocytes. An electrocardiogram was indicative of right ventricular hypertrophy.

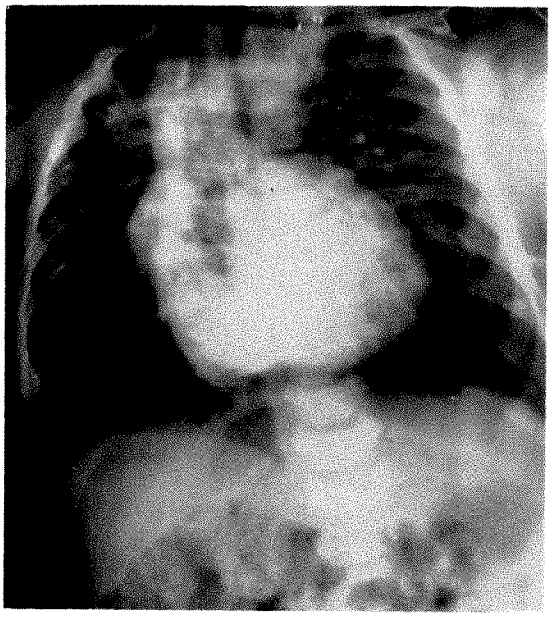


FIG. 1. Case I. Frontal angiocardiogram showing a single large atrium and a single large ventricle with simultaneous filling of the pulmonary arterial circulation and a dextroposed aorta.

On January 26, 1956 pneumoperitoneum (Fig. 2) and laminagraphy of the abdomen confirmed the clinical suspicion of an absent spleen. On February 19, 1956 he developed a left otitis media and thereafter he continued to have recurrent ear infections. Cardiac catheterization was carried out on March 22, 1956 and showed a 75 mm. Hg pressure gradient from right ventricle to pulmonary artery. The "right" ventricular systolic pressure was identical to the brachial artery systolic pressure. The catheter entered a superior right pulmonary vein from the "right" atrium.

Chest roentgenograms showed a large heart shadow which increased in size between January 23, 1956 (Fig. 3*A*) and October 10, 1957 (Fig. 3*B*). Upward tilting of the apex, a right sided aortic arch, a concave pulmonary artery segment, diminished pulmonary vascularity, symmetry of the major bronchi and a bifid right fourth rib were also demonstrated.

He subsequently had three hospital admissions for pneumonia and died on October 9, 1959. Autopsy specimens showed cor biloculare (failure of the interauricular and interventricular septa to develop). The heart and lungs together weighed 585 gm. (normal weight 225 gm.). The superior and inferior vena cava and the pulmonary veins entered a common auricle which was dilated and hypertrophied. A right

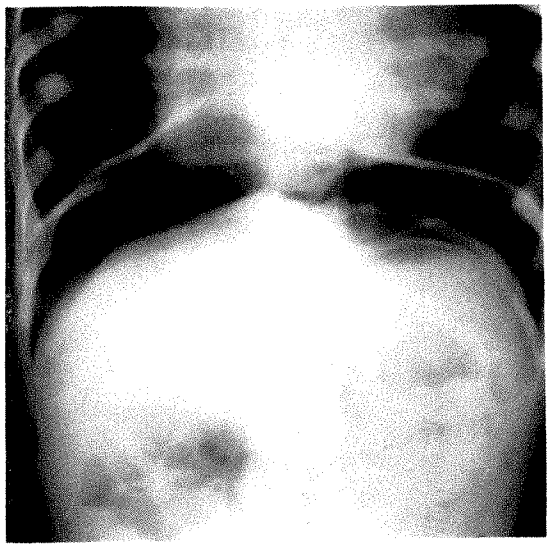


FIG. 2. Case 1. Angiocardiogram in the upright position after pneumoperitoneum showing a normal diaphragm and no evidence of splenic shadow. This was confirmed by multiple projections and laminagraphy.

auricular appendage was present anterolaterally and a left auricular appendage was present posteriorly. A wide muscular band extended from the right lateral wall of the auricle to the posterior base of the valve between the auricle and ventricle (Fig. 4*A*). The valve was 12 cm. in circumference and had three thickened leaflets with thickened cordae tendinae. The medial leaflets had three poorly defined divisions. The wall of the dilated common ventricle was hypertrophied and the endocardial surface was thickened. A common outflow tract led to the aorta anteriorly and the pulmonary artery posteriorly (Fig. 4*B*). The aortic valve was 4.5 cm. in circumference and the sinuses of Valsalva were dilated. The pulmonary artery was 2.5 cm. in circumference.

There was anomalous origin of the vessels arising from the aortic arch. A right subclavian and right common carotid artery arose from the right side of the aortic arch and an innominate artery from the left side. There was abnormal lobation of the lungs. In the left lung

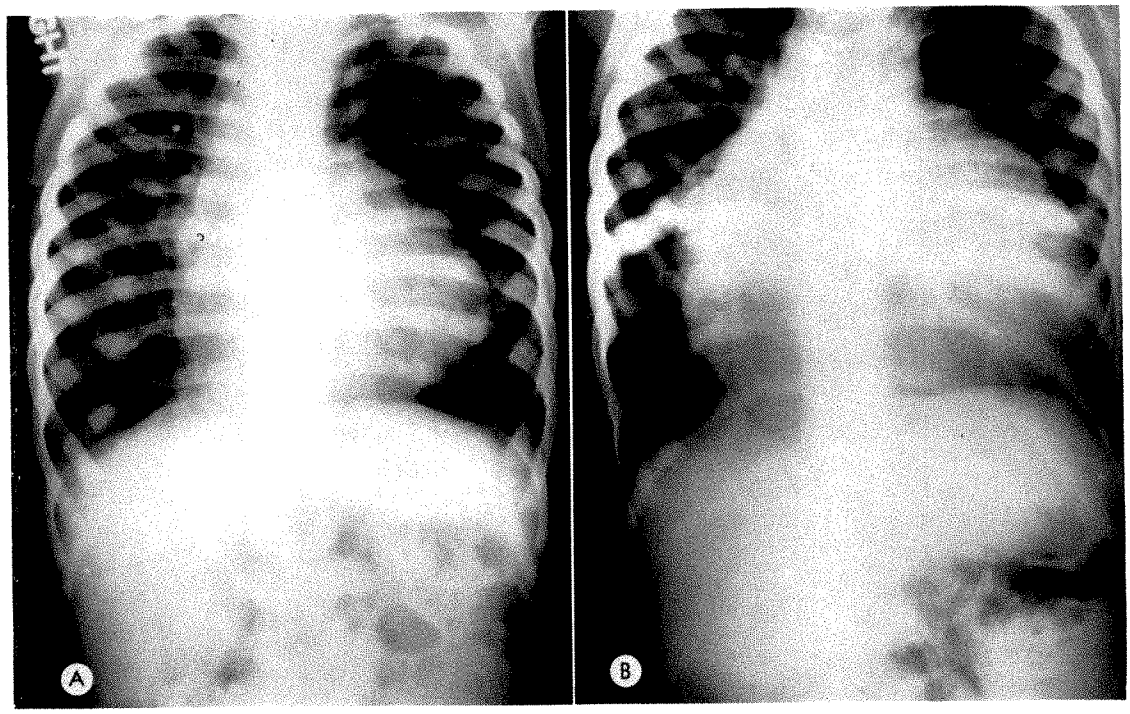


FIG. 3. Case 1. Teleroentgenograms of the chest. (*A*) Examination on January 23, 1956 showing enlargement of the cardiac shadow, upward tilting of the apex, concavity of the pulmonary artery segment and hypovascularity of the lung fields. There is a congenital anomaly of the anterior aspect of the right fourth rib and a right aortic knob. (*B*) Examination on October 10, 1957 showing increase in the size of the cardiac shadow. There is symmetry of the right and left bronchial tree.

three secondary bronchi and three main lobes were present. A small lobe was attached to the left apex and two extra lobes adjoined the left lower lobe. In the right lung, a small lobe was attached to the right apex and an extra lobe adjoined the right lower lobe.

The spleen was absent. There was acute and chronic bronchitis with bronchiectasis and emphysema. There also was incomplete rotation of the large bowel (cecum mobile). The cecum lay on the left side and the entire large bowel was free in the abdominal cavity; its mesentery was attached to the posterior abdominal wall in the midline.

Case II. This three month old white girl was the product of a normal full term delivery following an uneventful antenatal course. She was noted to have intermittent cyanotic attacks and a heart murmur and was admitted for study.

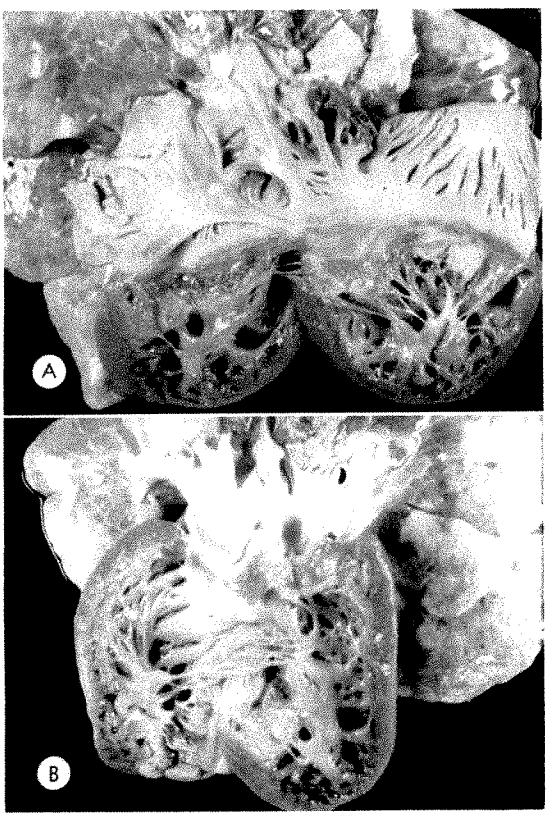


Fig. 4. Case I. Autopsy specimens showing cor biloculare. (*A*) A common auricle and a common ventricle are clearly seen. Note the muscular band extending from the right lateral wall of the auricle to the posterior base of the valve between the auricle and ventricle. (*B*) The pulmonary artery and aorta arise from a common ventricular outflow tract.

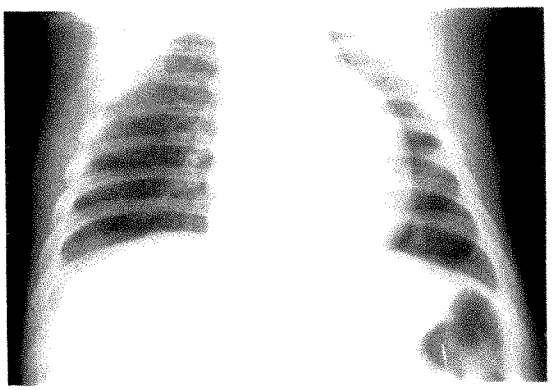


Fig. 5. Case II. Teleroentgenogram of a three month old female showing an enlarged heart shadow, a widened superior mediastinum, clear lung fields and a right sided stomach.

A chest roentgenogram (Fig. 5) showed a large heart with a wide superior mediastinum. A straight left border of the mediastinal shadow indicated a bilateral superior vena cava. Lung fields showed diminished vascularity. The right sided stomach shadow indicated partial situs inversus.

Physical examination disclosed a well developed cyanotic white infant. There was tachycardia and a systolic high pitched murmur loudest in the second and third intercostal space over the sternum. No thrill was felt. The abdomen was soft and slightly protuberant with the liver edge 2 cm. below the right costal margin.

Laboratory studies showed a blood hemoglobin of 16 gm. per cent; the white blood cell count was 12,800 with 51 per cent lymphocytes, 8 per cent monocytes and 41 per cent polymorphonuclear leukocytes. An electrocardiogram showed left axis deviation and left ventricular hypertrophy.

Her cyanotic attacks increased in frequency and severity with gradual deterioration. She died at three months of age.

Autopsy revealed a bilocular heart with incomplete partitioning of the atria, a single ventricle, a common atrioventricular canal, pulmonic stenosis, bilateral superior vena cava and a right aortic arch (Fig. 6 *A*).

The heart was moderately enlarged. A right superior vena cava and the inferior vena cava entered the right portion of the atrium. The

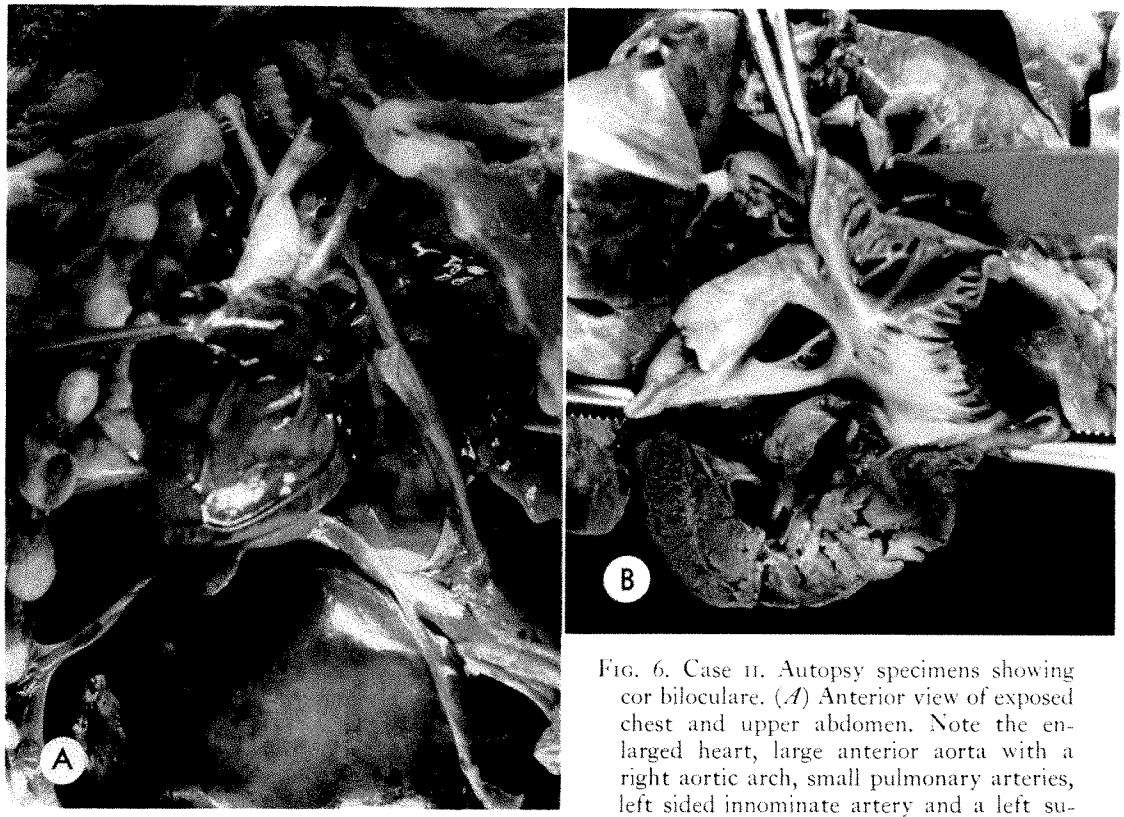


FIG. 6. Case II. Autopsy specimens showing cor biloculare. (*A*) Anterior view of exposed chest and upper abdomen. Note the enlarged heart, large anterior aorta with a right aortic arch, small pulmonary arteries, left sided innominate artery and a left superior vena cava. The spleen was absent. (*B*) Heart opened to show incomplete septation of the atria and a large, hypertrophied single ventricle. There is a common atrioventricular valve with four leaflets.

pulmonary veins and a left superior vena cava entered the left portion of the atrium. A 1 cm. defect, apparently representing the ostium secundum was evident in the upper portion of the interatrial septum; the inferior portion was absent. A common atrioventricular valve with 4 leaflets was present. There was no interventricular septum, but a large leaflet of the atrioventricular valve divided the ventricle into 2 chambers (Fig. 6 *B*). One chamber communicated with the atrium; the other communicated with the pulmonary artery and aorta.

The pulmonic valve was stenotic and had two thick small cusps. The aorta was of normal caliber, anterior and to the left of the pulmonary artery. There was a right aortic arch. The innominate artery passed to the left side of the neck, and the right common carotid and subclavian arteries orginated as separate trunks from the arch of the aorta.

Absence of the spleen and dextroposition of the stomach were noted. The entire colon was in the left lower abdomen. There was symmetric lobation of the lungs; each had three lobes.

CASE III. This female was first admitted at the age of twenty on January 19, 1950 with a history of heart trouble all her life beginning with dyspnea and a heart murmur in infancy. She was first noted to be cyanotic at the age of one week. She had been subject to bronchitis and repeated episodes of pneumonia.

She developed a severe pneumonia four years prior to admission and was hospitalized at another hospital. At that time she had severe shortness of breath and ankle swelling. She was digitalized and subsequently was able to continue normal activity until six months before her admission when she again noted increasing shortness of breath and ankle edema.

Examination showed a well developed woman with cyanotic lips. The apex cardiac impulse was in the fifth intercostal space in the left anterior axillary line. There was a systolic thrill, maximal over the sternum. Auscultation revealed a rough and loud systolic murmur over the entire precordium, loudest in the left parasternal area. A soft and blowing diastolic murmur was audible over the precordium, best heard along the sternal border. There was nor-

mal sinus rhythm. She had marked clubbing of her fingers and toes with cyanotic nailbeds.

Laboratory studies showed a blood hemoglobin of 25.5 gm. per cent and a red blood cell count of 7.5 million. Hematocrit was 72 per cent, and the white blood cell count was 9,600 with a normal differential. An electrocardiogram showed a bundle branch block.

A roentgenogram of the upper abdomen (Fig. 7) disclosed dextroposition of the stomach with partial transposition of the liver. A chest roentgenogram (Fig. 8) showed an enlarged heart and normal pulmonary vascularity. Angiocardiograms (Fig. 9, *A* and *B*) revealed a left superior vena cava and a large posterior atrium and ventricle. There was simultaneous filling of a right aortic arch and the pulmonary artery. Cardiac catheterization showed a right superior vena cava and no distinction between the right and left atrium. There was "right" ventricular hypertension.

She did well for two months following discharge but then was re-admitted because of respiratory distress and fever of unknown origin. She died on September 22, 1950.

Autopsy showed multiple congenital anomalies. Cor biloculare with a common outflow chamber, dextroposition of the aorta, and a persistent left superior vena cava were present.

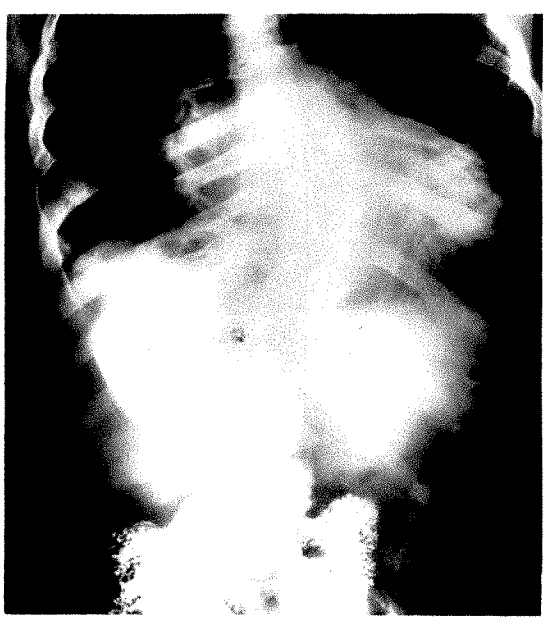


FIG. 7. Case III. A roentgenogram of the lower chest and upper abdomen after barium ingestion showing a right sided stomach and a centrally placed small intestine. Note the central position of the liver and the left dorsal scoliosis.

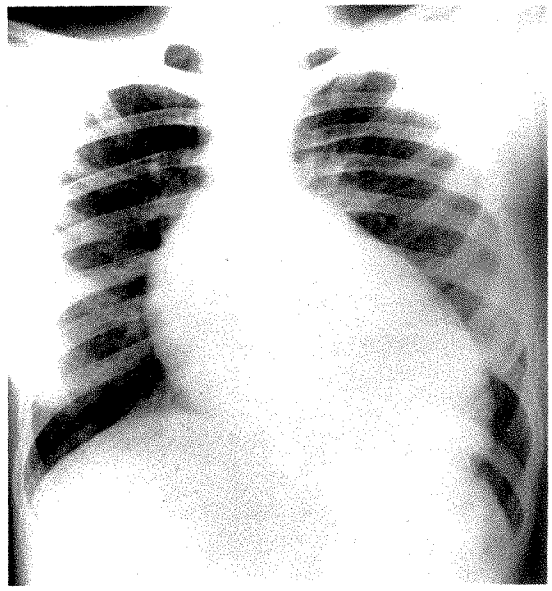


FIG. 8. Case III. Teleroentgenogram of the chest shows an enlarged heart shadow. The superior mediastinum is narrow with straight borders. The lung vascularity is within normal limits.

A single large auricle had a small rudimentary septum hanging from the middle of its anterior wall. Four pulmonary veins emptied into the right side of the auricle, while the inferior vena cava and the two superior venae cavae entered the left side of the chamber. There were two openings leading from the auricle: a well formed tricuspid valve led into a large ventricle, the wall of which was 10 mm. thick. A small slot-like opening at the left inferior position of the auricle suggesting a rudimentary mitral valve but devoid of any cusps led into a small outflow chamber situated in the left upper aspect of the heart. The large ventricle also communicated with the outflow chamber through a cuspless opening situated medially behind the tricuspid valve. Both the aorta and the pulmonary artery originated from this outflow chamber, the aorta anterior to the small pulmonary artery. The pulmonic valve was bicuspid. The aorta had a right sided arch, but was otherwise normal except for moderate atheromatosis. The two coronary arteries arose side by side from the posterior sinus of Valsalva.

There was partial situs inversus. The liver was located centrally in the upper abdomen. The esophagus traversed the right side of the diaphragm and the stomach was on the right side with the duodenum on the left. The spleen was absent.

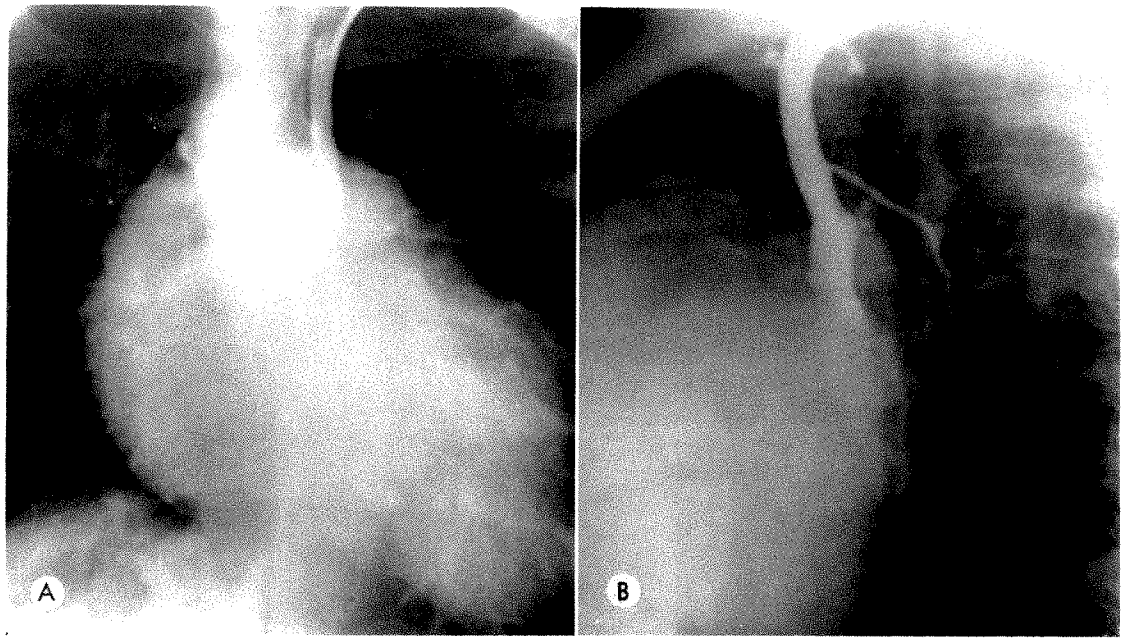


FIG. 9. Case III. (*A* and *B*) Angiocardiograms in frontal and lateral projections showing a left superior vena cava. There is a large posterior atrium and a large ventricle (cor biloculare). A right aortic arch and the pulmonary artery opacified simultaneously.

CASE IV. This two month old Negro female was admitted on December 17, 1949 because of dyspnea and cyanosis. She was born on October 11, 1949 at another hospital and was the 6 pound 11 ounce product of a full term spontaneous delivery. There were no complications during either pregnancy or delivery. The mother was told of no abnormality in the child at the time of discharge.

At about the age of three weeks the baby was examined by her physican and was said to have a heart murmur. The baby's lips were sometimes blue, especially when crying. When two months old the infant developed a "cold" with heavy breathing and an occasional cough. Breathing was rapid and the infant seemed very restless, with several episodes of vomiting.

Examination on admission showed a well developed and well nourished acutely ill infant with dyspnea and flaring alae nasi. The lips, nails and mucous membranes were cyanotic. The lungs were clear to percussion and auscultation. There was a thrill over the right parasternal area in the third and fourth interspaces. There was a loud harsh systolic murmur in the second to fourth intercostal spaces, transmitted over the whole precordium into the neck and the right axilla. The liver edge was 3 cm. below the right costal margin, extending across to the left axillary line, and slightly further below the costal margin on the left. The fingers and toes were cyanotic and clubbed.

Laboratory studies showed a blood hemoglobin of 17 gm. per cent; a red blood cell count of 5,300,000; and a white blood cell of 9,000. An electrocardiogram showed right axis deviation and tracings compatible with a true dextrocardia.

A roentgenogram of the chest and abdomen (Fig. 10) revealed a large heart which nearly filled the thoracic cavity. The liver was bilaterally symmetric, lying transversely across the entire upper half of the abdomen. Fluoroscopy showed dextrocardia and dextroposition of the stomach.

She was placed in an oxygen tent and treated with penicillin and digitalization. She showed no improvement and expired on the third day.

Autopsy showed multiple congenital anomalies with incomplete situs inversus. There was true dextroposition of the heart and aorta, and the stomach, duodenum and pancreas lay on the right side of the peritoneal cavity in the mirror image of their normal positions. The left lobe of the liver was unusually large but the gallbladder lay to the right side of the midline. The cecum and sigmoid were in normal position.

There was cor biloculare: the heart was hugely dilated and filled almost the entire thoracic cavity. It had two chambers with a large single auricle and single ventricle. The pulmonary veins and both venae cavae entered the large atrium in approximately normal position; the aorta and pulmonary artery both arose from the single ventricle. The coronary arteries and the branches of the aortic arch were in normal position. The valve between the two chambers appeared to have four cusps. The ductus arteriosus was completely closed. Histologically, the myocardium was normal, but there was congestion of liver and lungs. No trace of splenic tissue could be identified.

Case v. This male infant was born of an uncomplicated pregnancy on November 2, 1944. Cyanosis was first noted when he was three and one-half hours old and he was given oxygen without significant improvement. Physical examination revealed extreme cyanosis and severe dyspnea. There was slight dullness and diminution of breath sounds in the left upper lobe. The heart rate was rapid and a murmur was not detected.

Fluoroscopy of the chest revealed that the heart was moderately enlarged and globular. The lungs were clear. An electrocardiogram revealed left axis deviation. Laboratory studies showed a hemoglobin of 18.5 gm. per cent; a 5.0 million red blood cell count; and a white blood cell count of 22,700. There were 60 normoblasts per 100 white blood cells.

The baby expired on his second day of life and autopsy showed multiple congenital anomalies. There was cor biloculare with an incomplete fibrous septum between the right and left auricles. The part of the common chamber corresponding to the left auricle was about one-third the size of the right. There was no interventricular septum. Only one valve separated the auricle and ventricle and this had two cusps. A single large papillary muscle arose in the left posterior wall of the ventricle and was inserted at the base of the aorta. The ventricle emptied into the aorta, which was wider than normal. There were three normal aortic cusps and the coronary artery orifices were normally situated. The pulmonary artery was to the left and behind the aorta. Its proximal portion was atretic; distally the lumen was much smaller than that of the aorta. The ductus arteriosus was patent. There were only two arteries originating in the aortic arch, except for the ductus arteriosus. There were three lobes in each lung and congenital absence of the spleen.

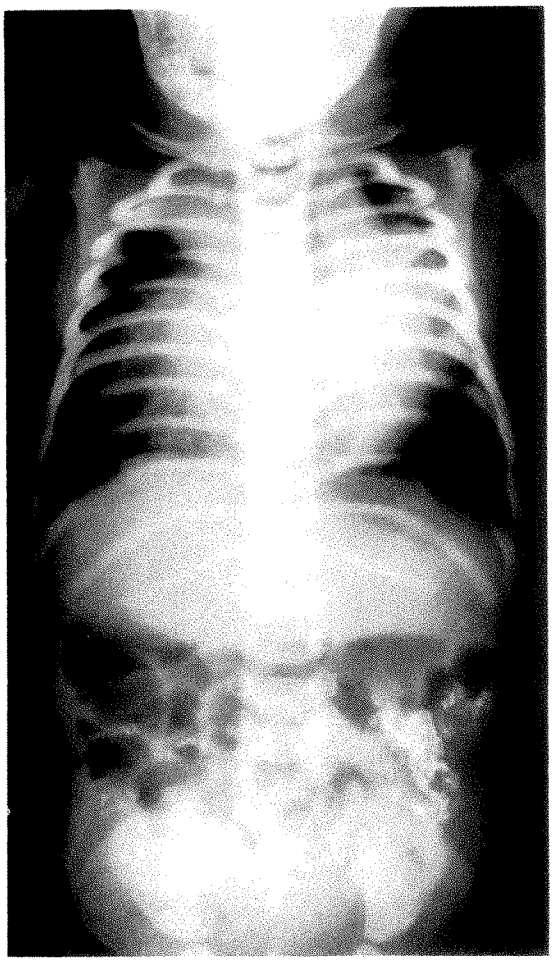


Fig. 10. Case iv. A roentgenogram of the chest and abdomen in a two month old female with asplenia and cor biloculare. There is an enlarged heart with dextrocardia. Note the symmetric liver shadow across the upper abdomen.

Clinical Findings

The first 2 cases of this syndrome to be diagnosed clinically were reported in 1952.[5] The diagnosis was made because of Heinz bodies in the erythrocytes of the peripheral blood in cyanotic infants. Bush and Ainger[4] point out that Heinz bodies are not found invariably in asplenia. However, there may be hematologic abnormalities similar to those found after splenectomy: target cells, decreased osmotic fragility, Howell-Jolly

bodies, transient normoblastemia, siderocytosis and leukocytosis. Thus, the blood picture in splenic agenesis is sufficiently specific to permit the antemortem diagnosis even in the absence of associated anomalies.[4]

The prognosis in this syndrome is extremely poor.[1,10,13,15] A severe anomaly of the heart and great vessels may be predicted and it may be expected that the benefit from surgical correction will be limited.[1] The awareness of agenesis of the spleen is valuable to the cardiovascular surgeon, since the cardiac anomalies often follow a predictable pattern. Aguilar *et al.*[1] reviewed 27 cases of splenic agenesis reported in the literature in addition to 3 of their own cases and were able to show a characteristic association of multiple congenital anomalies: (a) septal defects particularly atrioventricularis communis; (b) anomalous venous return to the heart; (c) pulmonary stenosis or atresia; (d) transposition of the great vessels; (e) accessory lobes of the lungs; (f) partial situs inversus of the abdominal viscera; and (g) abnormal mesenteric attachments.

Thus, all patients with undiagnosed cyanotic heart disease of infancy should have appropiate hematologic studies.[4,5] Abnormalities of the peripheral blood indicative of absence of the spleen are highly suggestive of the complete syndrome being present. Should a partial situs inversus of the abdominal viscera or symmetry of the liver or bronchial tree be evident with the hematologic abnormalities, the associated congenital heart disease is likely to be cor biloculare or a related variant.[4,5,12,14,18,19] Less frequently, there will also be abnormalities of the great vessels. Definitive diagnosis can be aided by roentgenographic studies including angiocardiography and gastrointestinal series.

Splenectomy in the infant before the age of six months is a more hazardous procedure from the standpoint of susceptibility to infection than in older children.[12,16] Serious infections in cyanotic infants, even without other evidence of heart disease, should suggest coexistent asplenia.[12]

ROENTGEN FINDINGS

By definition, the spleen is absent in all cases.[11] Absence of the splenic contour can be difficult to evaluate as the splenic shadow may not be seen in many normal subjects. Brogdon and Crow[3] noted that the lower pole of the spleen may be visualized on the anteroposterior roentgenogram of the abdomen in 44.4 per cent of adults. They feel that nonvisualization is not to be regarded as significant. In our Case 1, pneumoperitoneum and laminagraphy were utilized to document asplenia.

The liver, in this syndrome, is often transposed to the midline or left side of the abdomen with the larger lobe in the left upper quadrant. Ivemark[10] found that the liver was transposed to the left side in 17 of the 69 cases he studied. The liver is frequently symmetric, the left lobe equal to the right in size. This was noted in 31 per cent of the 69 cases of Ivemark.[10] The stomach was transposed to the right upper quadrant in 33 of these cases. Incomplete rotation of the bowel is not uncommon.[10,14,15]

Abnormal lobation of the lungs is frequent in this syndrome. It was evident in 46 of Ivemark's cases. Most commonly, there is symmetric lobation, the lungs being mirror images of each other, with three more or less complete lobes in each lung.[2,10]

The most frequent anomalies in the cardiovascular system are dextrocardia, right aortic arch, anomalies of the atrioventricular region, septal defects, anomalous connections of pulmonary veins and anomalous connection of systemic veins. These may be manifest on routine roentgenograms of the chest. Decreased pulmonary blood flow may also be evident.

Angiocardiography will allow visualization of cardiac chambers and document the size and position of the great vessels, anomalous shunting of the blood stream and anomalous venous return to the heart.[7] Cases of agenesis of the spleen with pulmonary stenosis or atresia with or without transposition of the great arteries may mimic an uncomplicated tetralogy of Fal-

lot.[10] The angiocardiographic findings of cor biloculare are often difficult to interpret. The visualization of a single atrium and a single ventricle on intravenous angiocardiography is compatible with cor biloculare, but other chambers or large septal defects can often not be excluded with certainty. Undoubtedly, selective angiocardiography is preferable in these patients.

SUMMARY

1. Five new cases of asplenia associated with other anomalies are presented with clinical, roentgenographic and autopsy data.

2. The frequency of partial situs inversus, tendency towards symmetry of lung and liver, and the absence of central septa of the heart (cor biloculare) in association with congenital absence of the spleen confirm the value of Ivemark's term: "asplenia, a teratologic syndrome of visceral symmetry."

3. Routine roentgenographic studies are valuable in diagnosis. Partial situs inversus, congenital cyanotic heart disease, symmetry of the bronchial tree and a centrally placed liver should suggest the syndrome. Special studies such as angiocardiography and pneumoperitoneum are presented for their value in antemortem diagnosis.

Nathaniel Finby, M.D.
St. Luke's Hospital
421 W. 113 Street
New York 25, New York

REFERENCES

1. Aguilar, M. J., Stephens, H. B., and Crane, J. T. Syndrome of congenital absence of spleen with associated cardiovascular and gastroenteric anomalies. *Circulation*, 1956, *14*, 520–531.
2. Brandt, H. M., and Liebow, A. A. Right pulmonary isomerism associated with venous, splenic, and other anomalies. *Lab. Invest.*, 1958, *7*, 469–504.
3. Brogdon, B. G., and Crow, N. E. Observations on normal spleen. *Radiology*, 1959, *72*, 412–414.
4. Bush, J. A., and Ainger, L. E. Congenital absence of spleen with congenital heart diseaes; report of case with antemortem diagnosis on basis of hematologic morphology. *Pediatrics*, 1955, *15*, 93–100.
5. Conn, J. J., Clark, T. E., and Kissane, R. W. Cor biloculare; report of four cases. *Am. J. Med.*, 1950, *8*, 180–186.
6. Daniel, D. S. Unusual case of ectopic splenic tissue resembling third testicle. *Ann. Surg.*, 1957, *145*, 960–962.
7. Dotter, C. T., and Steinberg, I. Angiocardiography. Paul B. Hoeber Company, New York, 1951.
8. Gasser, C., and Willi, H. Spontane Innenkörperbildung bei Milzagenesie. *Helvet. paediat. acta*, 1952, *7*, 369–382.
9. Gilbert, E. F., Nishimura, K., and Wedum, B. G. Congenital malformations of heart associated with splenic agenesis; with report of five cases. *Circulation*, 1958, *17*, 72–86.
10. Ivemark, B. I. Implications of agenesis of spleen on pathogenesis of cono-truncus anomalies in childhood; analysis of heart malformation in splenic agenesis syndrome, with 14 new cases. *Acta paediat.*, 1955, *44*, Suppl. 104.
11. Leadbeater, N. A. G. Congenital absence of spleen. *Brit. M. J.*, 1957, *1*, 342.
12. Levy, D. Congenital malformation of heart and great vessels associated with congenital absence of spleen. *Arch. Dis. Childhood*, 1957, *32*, 131–134.
13. Lyons, W. S., Hanlon, D. G., Helmholz, H. R., Jr., DuShane, J. W., and Edwards, J. E. Cardiac Clinics cxlviii. Congenital cardiac disease and asplenia; report of seven cases. *Proc. Staff Meet. Mayo Clin.*, 1957, *32*, 277–286.
14. Polhemus, D. W., and Schafer, W. B. Absent spleen syndrome; hematologic findings as aid to diagnosis. *Pediatrics*, 1959, *24*, 254–257.
15. Putschar, W. G. J., and Manion, W. C. Congenital absence of spleen and associated anomalies. *Am. J. Clin. Path.*, 1956, *26*, 429–470.
16. Smith, C. H., Erlandson, M., Schulman, I., and Stern, G. Hazard of severe infections in splenectomized infants and children. *Am. J. Med.*, 1957, *22*, 390–404.
17. Torgersen, J. Developmental anatomy of heart and etiology of congenital heart disease. *Acta radiol.*, 1950, *33*, 131–146.
18. Torgersen, J. Genetic factors in visceral asymmetry and in development and pathologic changes of lungs, heart and abdominal organs. *Arch. Path.*, 1949, *47*, 566–593.
19. Towers, B., and Middelton, H. Congenital absence of spleen associated with malformations of heart and transposition of viscera. *J. Path. & Bact.*, 1956, *72*, 553–560.

EPIDERMOID CYSTS OF THE SPLEEN IN CHILDREN*

By R. PARKER ALLEN, M.D., *and* VIRGIL R. CONDON, M.D.
DENVER, COLORADO

EPIDERMOID cysts of the spleen are rare. They are large fluid-filled masses with a lining of squamous epithelium and are thought to result from developmental rests. The diagnosis may be suspected when gross splenomegaly is encountered in a well child. Roentgen findings include a left upper quadrant mass and displacement or compression of the left kidney. Three cases are presented in which these effects upon the kidney are demonstrated.

REVIEW OF THE LITERATURE

Incidence. Andral, in 1829, wrote the first report of a splenic cyst. By 1956 Posener and Litherland[8] were able to collect 211 splenic cysts of all types from the literature. One of the earliest reports of epidermoid cysts was made by Shawan[10] in 1933. He had 1 case and collected 3 others. Bell,[1] in 1953, found 11 epidermoid cysts in a total of 188 cysts of the spleen which he reviewed. There are numerous other series reported in which a similar incidence of epidermoid cysts is found.[3,6,9]

Classification. There is some variation in the classification of splenic cysts but most authors have followed that of Fowler[3] who divides them into primary or true cysts with a well-defined epithelial or endothelial lining, and secondary or false cysts with no lining.

In the early classifications, true cysts included congenital, traumatic, inflammatory, parasitic and neoplastic forms. The false group included traumatic, degenerative and inflammatory cysts. Cellular metaplasia may account for the presence of a lining in some of the acquired conditions, so that traumatic and inflammatory cysts appear in both categories. The neoplastic group included epidermoids, dermoids, lymphangiomas, and cavernous hemangiomas, although some of these are perhaps more properly developmental aberrations. It is evident that a given case may be difficult to classify under this system. This accounts for the variations in the reported series.

Pathology. Epidermoid cysts are large and may contain from a few hundred to several thousand cubic centimeters of fluid. They are always lined with stratified squamous epithelium which is from five to ten cells thick.[3] The lining is surrounded by a dense hyalinized fibrous tissue. The cysts are usually solitary and are smooth externally. The internal lining may be thrown into folds resembling trabeculations. The fluid varies in color from brown to green and is usually cloudy. There may be fat, cholesterol or calcific debris in the fluid. Dermal structures such as hair follicles and sweat glands are not found. The remainder of the spleen is normal except for pressure atrophy.

Pathogenesis. The true cause for the formation of epidermoid cysts has not been determined. Several theories have been proposed. Shawan[10] and Fowler[3] have suggested that a portion of the celomic cavity capable of forming epidermis is enclosed in the splenic anlage during fetal development. Transformation from splenic mesoderm and from lymphoid endothelium has also been mentioned by these authors. Posener and Litherland[8] further suggest metaplasia following trauma, infarction, hemorrhage, and infection.

CLINICAL MANIFESTATIONS

Although splenic cysts in general are most often seen in the twenty to fifty year age group, epidermoids are found more frequently in the group under twenty years.[5] They are rarely found under five years of age.

The symptoms are minimal. Left upper

* From the Department of Radiology, Children's Hospital, Denver, Colorado.

quadrant fullness with vague pain which may radiate to the shoulder, chest, or back may be present. As the mass enlarges, nausea, vomiting, constipation, and dyspnea may appear secondary to displacement of abdominal and thoracic contents. There is frequently a history of either recent or old trauma.

Kidney function may be disturbed by the presence of any large abdominal mass which causes ureteral obstruction, pressure on the kidney, or pressure on the renal artery. Hoffman[4] reported a splenic cyst associated with albuminuria that disappeared following splenectomy. In one of the following case reports, the presenting clinical findings were hypertension and albuminuria.

The physical findings are minimal except for palpation of the mass at examination of the abdomen. Because of their asymptomatic nature, splenic cysts are usually large at the time of discovery. The left upper quadrant mass is usually smooth, nontender, and moves with respiration.

ROENTGEN MANIFESTATIONS

PLAIN ROENTGENOGRAPHY

A large soft tissue mass of water density is seen in the left upper quadrant of the abdomen. The majority of cases seen in childhood do not show visible calcium; however, scattered flecks of calcium may be seen and occasionally the whole mass appears to be calcified. The stomach is displaced to the right. The splenic flexure of the colon is displaced downward and/or to the right. The psoas margin may or may not be obscured. In the lateral projection the mass is seen extending from the anterior wall to the posterior wall. There is frequently a rounded indentation on the anterior wall of the displaced stomach.[11] Contrast studies of the gastrointestinal tract may serve to better delineate these displacements. The left side of the diaphragm may be elevated and left lower lobar pneumonia, atelectasis, and basal pleural effusion have been described. As with any large abdominal mass, there may be limitation of motion of the left side of the diaphragm.

INTRAVENOUS UROGRAPHY

Intravenous urography is of paramount importance in the investigation of an abdominal mass in childhood. The urinary tract may be entirely normal. Frequently, there is either compression or displacement of the left kidney. Displacement is usually downward but it may be upward.[2] Forde *et al.*[2] list 26 cases of displacement of the left kidney by splenic enlargement. In 14, the displacement was downward; in 9, downward and medial; and in 2, upward. Whether displaced or not, the function is good and there is no intrinsic deformity of the calyceal system. Some ureteral obstruction from external pressure may be present with varying degrees of dilatation and persistence of filling. Other cases may show poor filling due to direct pressure or, possibly, due to pressure upon the vascular supply. This may be the mechanism of the hypertension which is seen in cases such as our Case III presented below.

DIFFERENTIAL DIAGNOSIS

Roentgenologically, the differential diagnosis includes other causes of splenic enlargement. Tumors such as lymphangioma and hemangioma are seldom as large. Acquired cysts are impossible to differentiate except by microscopic examination. The history, when definite, may be helpful. Generalized splenic enlargements are usually associated with acute infections or serious diseases such as leukemia and the reticuloses. In these cases the contour of the spleen is maintained with elongation being the most prominent feature. Parasitic cysts of the spleen are diagnosed by their geographic occurrence more than by their roentgen features, although calcification of the wall is a frequent finding.

The differential diagnosis also includes numerous masses outside the spleen. The only significant pancreatic mass in the age group under consideration is the pseudocyst. It is usually not as large and not

situated in as lateral a position and it tends to displace the stomach anteriorly and to the left. Omental and mesenteric cysts are usually movable and rarely are found so far to the left side. Tumors of the left lobe of the liver may closely simulate splenic cysts. They are not so large and frequently the displaced spleen can be seen. Retroperitoneal tumors, including neuroblastoma, usually show their site of origin in the lateral view, especially if skeletal structures are involved. Intrinsic renal tumors almost invariable distort the calyceal pattern and in both renal and pararenal tumors the normal spleen shadow is visible.

The left kidney is not displaced with pancreatic cysts, mesenteric cysts, and tumors of the liver. Neuroblastoma usually displaces the kidney downward when the site of origin is the adrenal gland but the kidney may be displaced upward when the origin is in the sympathetic chain below the kidney. Other retroperitoneal tumors may displace the kidney in any direction according to their site of origin. Retroperitoneal tumors, including neuroblastoma, frequently rotate the kidney as well as displace it, while displacement from splenic enlargement is usually without rotation.

REPORT OF CASES

CASE 1. W.N., an eleven year old boy, was admitted with a complaint of pain in the left hypochondrium. The pain had started about ten days before. It was constant, although occasionally cramping, and was not related to position or eating. There were no other complaints and the past history was negative.

On examination the patient appeared well. The pulse, temperature, and respirations were normal. The blood pressure was 110/55. Physical examination was negative except for the abdomen where there was a 15 by 10 cm. mass in the left upper quadrant extending down to the umbilicus. The mass was smooth, nontender, and fixed, except for motion with respiration. There was no clinical evidence of peritoneal fluid or of peritoneal irritation. Laboratory tests were within normal limits.

Roentgen examination revealed a homogeneous mass of water density in the left upper quadrant extending down to the iliac crest. The margins were not well defined but the mass displaced the colon, small bowel, and stomach to the right side of the abdomen. The margins of the liver, spleen, and psoas muscle were not distinct. Except for displacement, the bowel pattern was normal. Intravenous pyelogram (Fig. 1) revealed good function bilaterally and an anatomically normal right side. The left kidney was displaced upward so that the lower pole was at the level of L-1. There was no intrinsic deformity of the calyceal system. The ureter was well filled. The early studies showed a nephrogram, suggesting partial obstruction of the left ureter.

At laparotomy the mass was found to be a large cyst of the spleen which contained 1,149 cc. of rust colored fluid. It was removed.

Examination of the specimen (Fig. 4 *A*) revealed a large cyst with the wall composed of

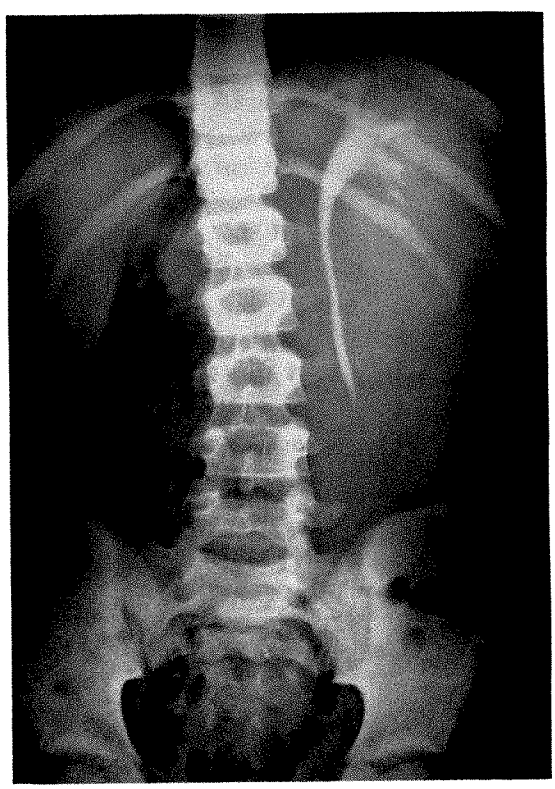


FIG. 1. Case 1. Intravenous pyelogram shows the large left upper quadrant mass displacing the stomach and bowel to the right and downward. The left kidney is displaced upward so that the upper pole lies just under the diaphragm. There is slight compression of the left ureter.

hyalinized collagen material lined by stratified squamous epithelium. There were no skin appendages present in the specimen so the pathologic diagnosis was epidermoid cyst of the spleen.

CASE II. M.N., an eight year old girl, was admitted with a complaint of swelling in the left side of the abdomen which had been present for one week. There was no pain or complaint of gastrointestinal disturbance. The child had always been in good health. One year previously she had been thrown from a horse but there were no abdominal complaints at that time.

The only abnormal physical finding was a large firm mass occupying the left upper quadrant of the abdomen and measuring 13 by 14 cm. It was smooth and nontender. There was no evidence of peritoneal irritation or free fluid. Temperature, pulse, respirations, and blood pressure were normal. Routine laboratory examinations were normal.

A roentgenogram of the abdomen (Fig. 2) revealed a mass of water density occupying the left upper quadrant displacing the colon downward and the stomach to the right. There was no evidence of free peritoneal fluid and the gas pattern of the bowel was normal except for displacement. The psoas margins were distinct. A definite splenic shadow was not visible. Intravenous urography revealed a normal kidney on the right side. On the left side the calyceal structures were not well filled and the kidney was displaced downward. There was no intrinsic distortion of the calyceal structures. The position of the mass relative to the kidney was such that the incomplete filling could have been the result of direct pressure.

At laparotomy the mass was identified as the spleen. It was cystic and brownish in color. The spleen including the mass was removed.

The specimen (Fig. 4 *B*) measured 20 by 15 by 10 cm. and weighed 1,725 gm. It consisted of a large cyst filled with a turbid greenish-brown fluid. Microscopic examination revealed a fibrous wall lined by stratified squamous epithelium. No accessory dermal structures were found and the diagnosis was epidermoid cyst of the spleen.

CASE III. J.B., a thirteen year old boy, was found to have hypertension and albuminuria on a routine physical examination. The patient

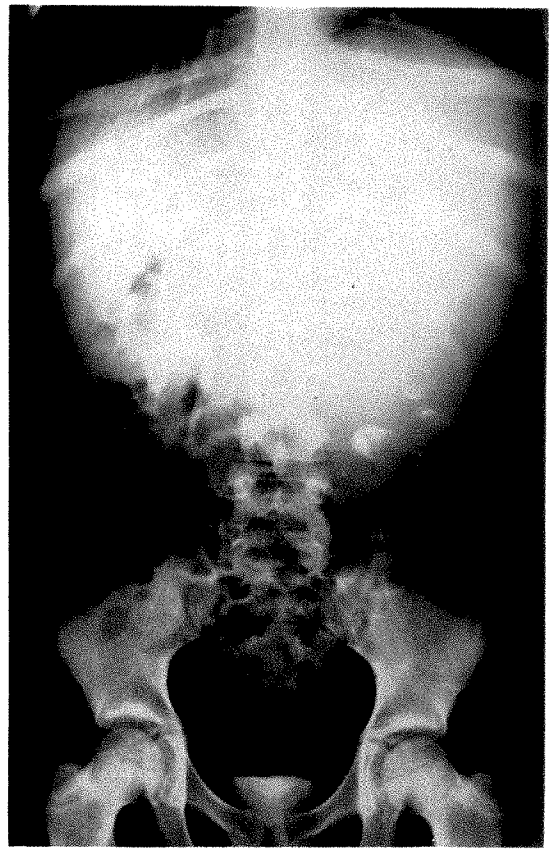


FIG. 2. Case II. The left upper quadrant mass has displaced the intestinal gas pattern downward and to the right and the kidney is displaced downward so that it lies over the iliac crest.

was well and had been unusually active athletically. There were no symptoms referable to the cardiovascular or urinary systems.

On admission to the hospital, the temperature and respirations were normal. The blood pressure ranged from 152/104 to 130/72 with most of the systolic readings over 140. The pulse rate ranged from 80 to 114. The remainder of the physical examination was normal except for the finding of a large mass in the left upper quadrant. It moved with respiration and was not tender. Laboratory work demonstrated 25 mg. per 100 mm.^{3} albumin in the urine and 50 mg. per 100 mm.^{3} on a second examination. There were occasional white blood cells, phosphate crystals and granular casts. Blood creatinine, nonprotein nitrogen and serum protein were within normal limits.

Roentgen examination of the abdomen (Fig. 3, *A* and *B*) revealed a large mass of soft tissue density in the left upper quadrant. It displaced

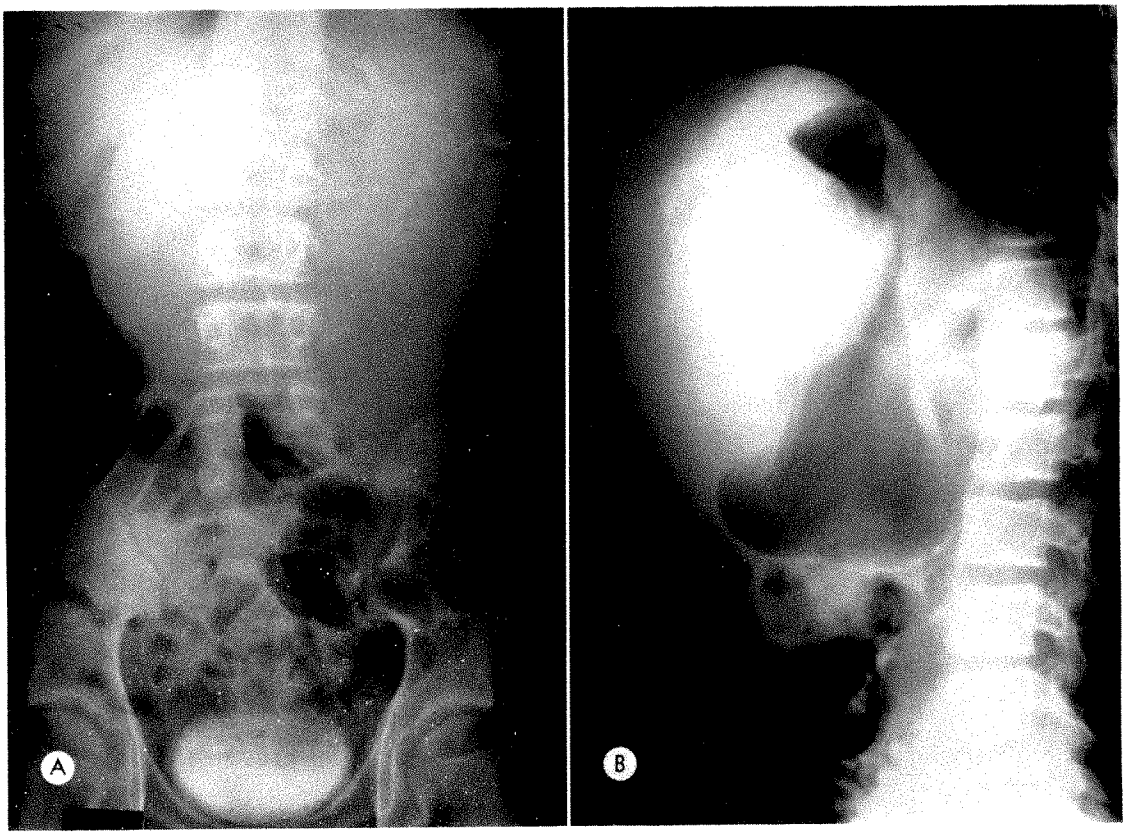


FIG. 3. Case III. (*A*) The left upper quadrant mass has displaced the stomach far to the right and the transverse colon downward. The splenic flexure is situated over the wing of the ilium. The left kidney is displaced slightly laterally and its excretory function appears suppressed by external pressure. (*B*) The lateral roentgenogram shows the indentation of the mass on the anterior margin of the stomach.

the gas-filled colon inferiorly and the stomach to the right. The underlying psoas shadow was visible as was the renal outline. The margin of a normal spleen was not demonstrated. No free fluid was seen. There was a minimal lumbar scoliosis concaved to the left. Intravenous urography showed a normal right kidney. On the left side the collecting system did not fill well but there was no intrinsic deformity and only slight lateral displacement of the kidney. On the lateral roentgenogram the mass was seen to be indenting the anterior wall of the gas-filled stomach.

Incomplete filling of the collecting system on the left side as compared to the good right kidney was quite impressive and was interpreted as indicating pressure of the mass on the kidney. It could, however, have been the result of pressure upon the renal artery. Lumbar aortography was not done.

At laparotomy the left upper quadrant mass was identified as the spleen. It was smooth and reddish-purple in color. After removal of the mass, it was noted that the left kidney appeared somewhat flattened where the mass had been pressing upon it.

Following operation, the blood pressure returned to normal, ranging from 134/70 to 120/64. Repeat urinalysis showed a normal specific gravity, no albumin and only an occasional white blood cell.

The pathologic specimen (Fig. 4 *C*), including spleen and cyst, weighed 3,050 gm. and the cyst contained 2,000 cc. of cloudy brown fluid. The spleen appeared congested. The cyst wall was thick and trabeculated. Microscopic examination showed that the wall was composed of dense fibrous connective tissue lined, for the most part, by a thin layer of stratified squamous epithelium. There were a few small foci of calcified debris. Pathologic diagnosis was epidermoid cyst of the spleen.

SUMMARY

Three children with epidermoid cysts of the spleen are reported. The typical patient is over five years of age and presents with an asymptomatic left upper quadrant mass. The complaints in one case were hypertension and albuminuria, probably due to the pressure of the mass on the kidney.

The roentgen features include the left upper quadrant mass with displacement of the gas-filled stomach and bowel, absence of the normal splenic margin, and displacement of the left kidney, usually downward but occasionally laterally and upward.

The history, physical findings and roentgen examination, when considered together, may be sufficiently characteristic to lead to the correct diagnosis.

Virgil R. Condon, M.D.
The Children's Hospital
Nineteenth Avenue at Downing
Denver 18, Colorado

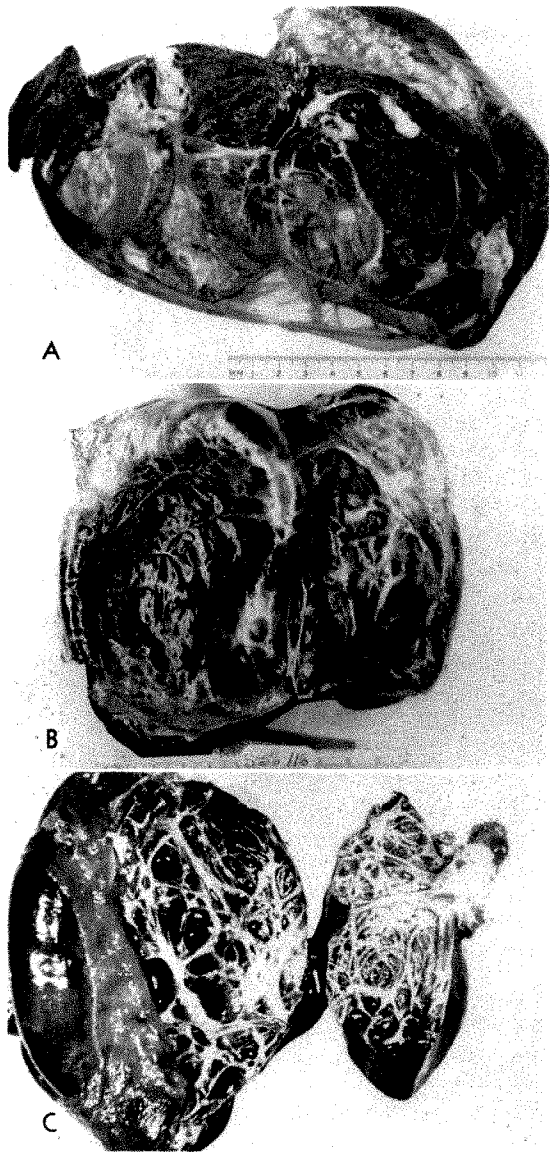


FIG. 4. The cut surgical specimens of (*A*) Case I, (*B*) Case II and (*C*) Case III show the trabeculated cyst wall and demonstrate a remarkable similarity.

REFERENCES

1. BELL, R. P., JR. Splenic cysts with report of case of large unilocular cyst of rapid growth. *Ann. Surg.*, 1953, *137*, 781–786.
2. FORDE, W. J., OSTROLENK, D. G., and FINBY, N. Renal displacement associated with enlargement of spleen. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *84*, 889–897.
3. FOWLER, R. H. Cystic tumors of spleen. *Internat. Abstr. Surg.*, 1940, *70*, 213–223.
4. HOFFMAN, E. Non-parasitic splenic cysts. *Am. J. Surg.*, 1957, *93*, 765–770.
5. LEE, R. E., and ARNSPIGER, L. A. Epidermoid cyst of spleen; report of case. *Arch. Surg.*, 1958, *77*, 10–12.
6. MARTIN, J. W. Congenital splenic cysts. *Am. J. Surg.*, 1958, *96*, 302–308.
7. MCCLURE, R. D., and ALTEMEIER, W. A. Cysts of spleen. *Ann. Surg.*, 1942, *116*, 98–102.
8. POSENER, L. J., and LITHERLAND, O. K. Epidermoid cyst of spleen. *Canad. M. A. J.*, 1956, *75*, 510–512.
9. SCHWARTZ, A. D. Traumatic cyst of spleen. *Am. J. Surg.*, 1955, *89*, 1084–1085.
10. SHAWAN, H. K. Epidermoid cysts of spleen. *Arch. Surg.*, 1933, *27*, 63–74.
11. SWEET, R. H. Single true cysts of spleen; report of 3 cases. *New England J. Med.*, 1943, *228*, 705–710.

BILATERAL HEMORRHAGIC PSEUDOCYSTS OF THE ADRENAL GLANDS IN A NEWBORN*

By ARCHIBALD C. WAGNER, M.D.
RICHMOND, VIRGINIA

THE purpose of this paper is to describe what is, to our knowledge, the youngest reported patient demonstrating roentgen signs of cystic lesions of the adrenal glands.

REPORT OF A CASE

This white female infant was born June 7, 1960 of a normal spontaneous delivery, with vertex presentation, and cried spontaneously. During the first three days of life, jaundice, with bilirubin reaching 20.3 mg. per cent, intermittent cyanosis and leukocytosis were noted. Roentgenograms of the abdomen and chest were unremarkable at that time. Antibiotics were started and a complete exchange transfusion was performed. Blood and cerebrospinal fluid cultures were negative and the infant improved.

Escherichia coli was cultured from the urine on the ninth day. In the twelfth day of life the infant vomited several times and developed profound shock and cardiac arrest which responded to intracardiac epinephrine and mouth to mouth breathing. A roentgenogram of the abdomen at this time revealed bilateral symmetric rim-like calcifications in both upper quadrants of the abdomen (Fig. 1, *A* and *B*). These were interpreted as most likely representing calcified cystic lesions of the adrenal glands or kidneys. Bilateral masses were now palpable in the upper abdomen. Intravenous pyelography the following day was technically poor and gave no significant information.

At the age of fourteen days, a retrograde pyelogram showed that the calcified masses were in the suprarenal areas, displacing the kidneys downward and distorting the right collecting system (Fig. 2). A lipiodol swallow demonstrated that the stomach and the duodenum were displaced anteriorly.

Exploratory laparotomy was performed on the fifteenth day of life and a cystic lesion containing old blood and measuring 7.5 cm. in diameter was removed from each adrenal gland (Fig. 3).

Microscopically, these cysts showed a

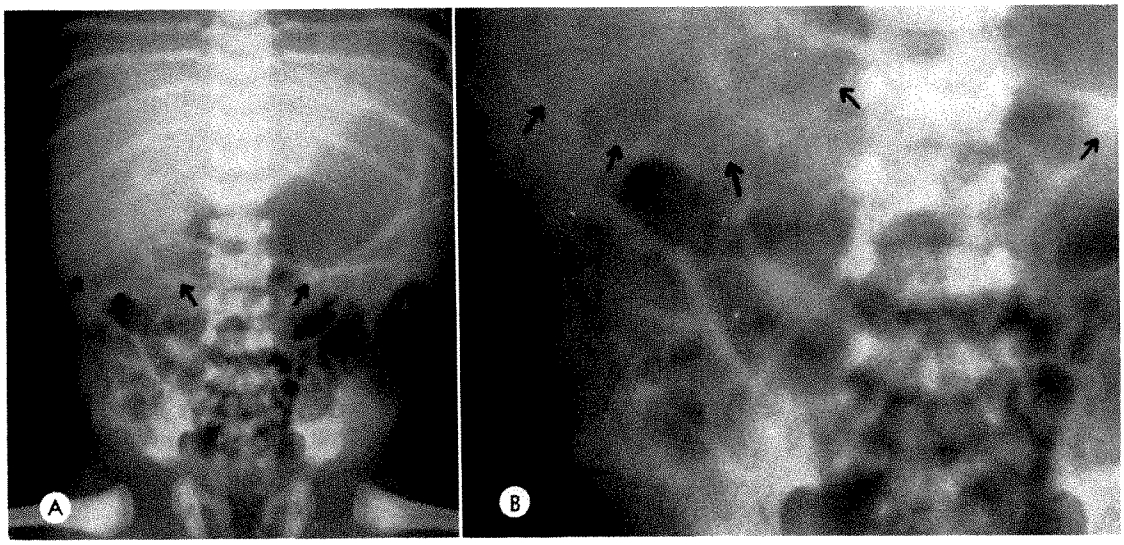


FIG. 1. (*A*) Plain abdominal roentgenogram of the infant at twelve days of age showing rim-like calcific densities (arrows) in both upper abdominal quadrants. (*B*) Detail of *A* showing the rim-like calcification (arrows) in the right upper quadrant.

* From the Department of Radiology, Medical College of Virginia Hospitals, Richmond, Virginia.

centrally located area of infarcted tissue and hemorrhage and a peripheral zone of intact adrenal tissue; between these two areas was a thin layer of calcified material (Fig. 4).

The final diagnosis was hemorrhagic pseudocysts of the adrenal glands. It was felt that the hemorrhage was secondary to infection.

The infant was discharged on August 19, 1960 on adrenal replacement therapy and was doing well. At the age of nine months she was still thriving except for intermittent bacilluria.

DISCUSSION

Adrenal cysts are uncommon. A review of the literature in 1958 disclosed 155 reported cases, found either at surgery or at autopsy, with patient age ranging from the newborn to seventy-six years.[1] In these 155 cases the most common type of cystic lesion encountered was pseudocyst formation incident to adrenal hemorrhage (50 cases).

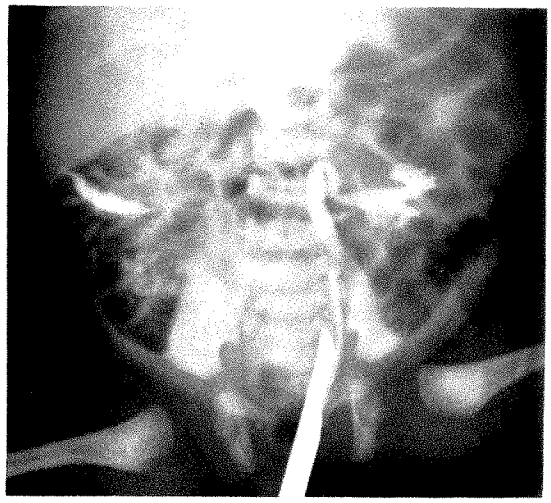


Fig. 2. Retrograde pyelogram of the infant at fourteen days of age showing both kidneys displaced inferiorly and rotated laterally by suprarenal masses. There is some distortion of the collecting system on the right.

In the young, adrenal hemorrhage is not rare. It was noted in slightly more than 1 per cent of a series of autopsies performed on infants and children,[33] and it may be unilateral or, more commonly, bilateral.[36] It has been reported in association with birth trauma (including forceful resuscitation),[18,32,33,36] infection,[18,32,33,36] hemorrhagic disease of the newborn,[32,33] physiologic involution,[18,36] regional suppuration with thrombosis of the adrenal vein,[20] burns,[33,36] hemophilia,[36] syphilis,[56] leukemia,[36] and incompatible blood transfusion.[33] Adrenal hemorrhage in the newborn period is most commonly due to trauma at the time of delivery.[32] The hemorrhagic mass may even reach such size as to cause obstruction of the colon from extrinsic pressure.[10]

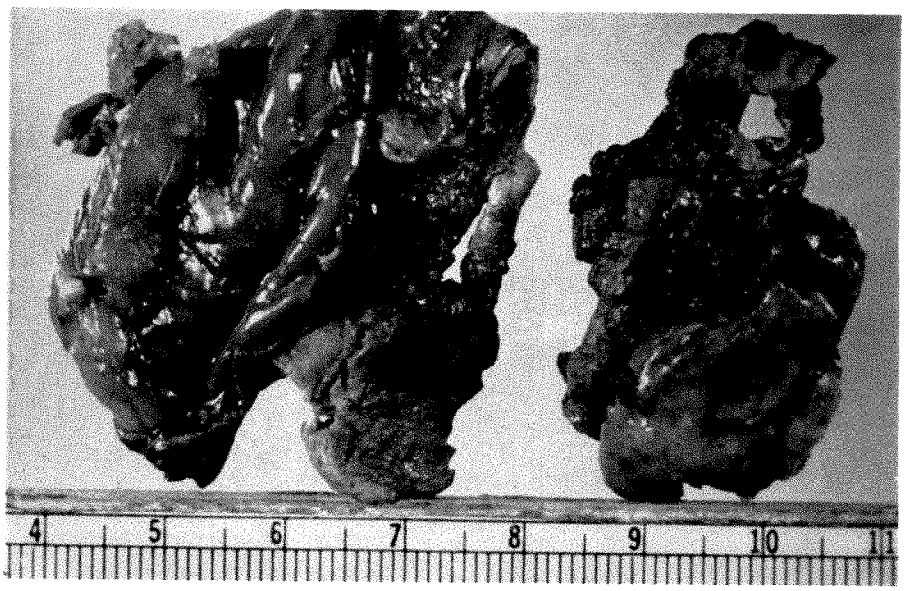


Fig. 3. Photograph of the surgical specimens showing part of each adrenal pseudocyst.

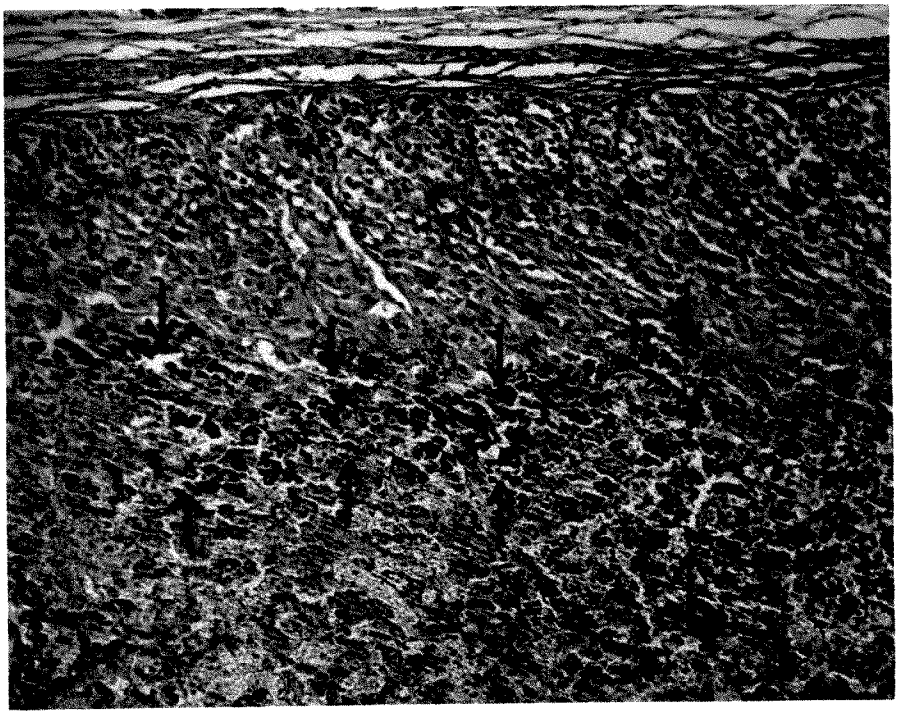


FIG. 4. Photomicrograph of a section of one pseudocyst showing a layer of calcification (arrows) between peripheral, normal adrenal gland tissue (above) and centrally located infarcted tissue and hemorrhage (below) (×100).

Calcification within the adrenal glands may occur following hemorrhage[11,29,34] as well as with tuberculosis,[4,8,11,29,35] ganglioneuroma,[11] neuroblastoma,[11,23] pheochromocytoma,[6,14] carcinoma,[11,31] adenoma,[3] pseudocysts,[21,22,25–28,30,39] generalized xanthomatosis,[22] Niemann-Pick's disease,[28] and *Echinococcus* cysts. The calcification has assumed a rim-like character, however, only in association with cystic lesions of various types, including cystic degeneration within a tumor.

Although the presence of adrenal calcification has been noted at autopsy as early as eight days following adrenal hemorrhage in the newborn,[33] the youngest patient previously reported with roentgenographic evidence of adrenal calcification was four months old.[11]

The mechanism of pathologic calcification has been adequately covered in previous reports,[5,38] as has the clinical picture of adrenal insufficiency.[12,13,15,17,18,24,32,33,36,37]

Calcified cystic lesions of the adrenal glands must be differentiated from other lesions in the abdomen and retroperitoneum which may also show rim-like calcification, such as aneurysms of various abdominal arteries, hepatic and splenic cysts,[28] renal cysts,[16] dermoid and *Echinococcus* cysts,[28] cysts of the mesentery,[19] pancreatic cysts, Wilms's tumor,[22] and renal carcinoma.[9] This is best accomplished by urography. However, with bilateral calcified adrenal cysts, especially in infancy, the diagnosis may be strongly suspected on the basis of the plain roentgenograms alone, since, of the above named lesions, only renal cysts show any tendency to be bilaterally located, and to our knowledge symmetric rim-like calcification has not been reported in this latter condition in early infancy.

SUMMARY

Hemorrhagic pseudocysts of the adrenal glands may show roentgenographically discernible rim-like calcification in a patient

as young as twelve days of age. With bilateral calcification on plain roentgenograms, this diagnosis should be the first consideration and can easily be confirmed by pyelography. This sign should be specifically looked for in any infant exhibiting unexplained gastrointestinal symptoms, collapse, irritability or other signs of adrenal insufficiency.

Department of Radiology
The Fauquier Hospital
Warrenton, Virginia

Appreciation is expressed to Dr. Helen L. Morton and Dr. Jack M. Harrison for permission to publish the roentgen features of this case and to Dr. Frederick B. Mandeville and Dr. Christian V. Cimmino for their advice and assistance in the preparation of this paper.

REFERENCES

1. ABESHOUSE, G. A., GOLDSTEIN, R. B., and ABESHOUSE, B. S. Adrenal cysts; review of literature and report of three cases. *J. Urol.*, 1959, *81*, 711–719.
2. ABRAMOV, A., SCHORR, S., and WOLMAN, M. Generalized xanthomatosis with calcified adrenals. *Am. J. Dis. Child.*, 1956, *91*, 282–286.
3. ANDERSON, M. X., ROBERTS, H. G., and SMITH, E. T. Calcified cyst of adrenal cortex without endocrine symptoms. *Radiology*, 1950, *54*, 236–241.
4. BALL, R. G., GREENE, C. H., CAMP, J. D., and ROWNTREE, L. G. Calcification in tuberculosis of suprarenal glands: roentgenographic study in Addison's disease. *J.A.M.A.*, 1932, *98*, 954–961.
5. BARR, D. P. Pathological calcification. *Physiol. Rev.*, 1932, *12*, 593–624.
6. BARTELS, E. C., and CATTELL, R. C. Pheochromocytoma: its diagnosis and treatment. *Ann. Surg.*, 1950, *131*, 903–916.
7. CAFFEY, J. Pediatric X-Ray Diagnosis. Third edition. Year Book Publishers, Inc., Chicago, 1956.
8. CAMP, J. D., BALL, R. G., and GREENE, C. H. Calcification of suprarenal glands in Addison's disease. AM. J. ROENTGENOL. & RAD. THERAPY, 1932, *28*, 594–597.
9. CANNON, A. H., ZANON, B., JR., and KARRAS, B. G. Cystic calcification in kidney; its occurrence in malignant renal tumors. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *84*, 837–848.
10. CORCORAN, W. J., and STRAUSS, A. A. Suprarenal hemorrhage in newborn. *J.A.M.A.*, 1924, *82*, 626–628.
11. DRUCKER, V., and RODRIGUEZ, C. E. Extensive bilateral calcification within adrenal hemorrhage. *Radiology*, 1955, *64*, 258–261.
12. DUDGEON, L. S. Etiology, pathology and diagnosis of adrenal hemorrhage. *Am. J. M. Sc.*, 1904, *127*, 134–146.
13. FARR, C. E., and SEMSROTH, K. Suprarenal hemorrhage. *Arch. Surg.*, 1927, *15*, 627–634.
14. FEIST, J. H., and LASSER, E. C. Pheochromocytoma with large cystic calcification and associated sphenoid ridge malformation. *Radiology*, 1961, *76*, 21–26.
15. GEPPERT, L. J., SPENCER, W. A., and RICHMOND, A. M. Adrenal insufficiency in infancy. *J. Pediat.*, 1950, *37*, 1–22.
16. GOLDSTEIN, A. E., and ABESHOUSE, B. S. Calcification and ossification of kidney. *Radiology*, 1938, *30*, 544–578.
17. GOLDZIEHER, M. A., and GORDON, M. B. Syndrome of adrenal hemorrhage in new-born. *Endocrinology*, 1932, *16*, 165–181.
18. HILL, E. E., and WILLIAMS, J. A. Massive adrenal hemorrhage in newborn. *Arch. Dis. Childhood*, 1959, *34*, 168–182.
19. HINKEL, C. L. Mesenteric cysts; their roentgen diagnosis. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *48*, 167–176.
20. LAVENSON, R. S. Acute insufficiency of adrenals. *Arch. Int. Med.*, 1908, *2*, 62–73.
21. LEVISON, P. Case of bilateral adrenal cysts. *Endocrinology*, 1933, *17*, 372–376.
22. MCAFEE, J. G., and BALLI, C. E. Radiological diagnosis of diseases of adrenal origin. *Am. J. M. Sc.*, 1956, *232*, 572–599.
23. MANDEVILLE, F. B. Sympathoblastoma (neuroblastoma), adrenal, abdominal, and mediastinal. *Urol. & Cutan. Rev.*, 1947, *51*, 448–452.
24. MOORE, F. P., II, and CERMAK, E. G. Adrenal cysts and adrenal insufficiency in infant with fatal termination. *J. Pediat.*, 1950, *36*, 91–95.
25. O'CONNELL, N. D. Adrenal cysts. *Brit. J. Radiol.* 1959, *32*, 490–492.
26. PALUBINSKAS, A. J., CHRISTENSEN, W. R., HARRISON, J. H., and SOSMAN, M. C. Calcified adrenal cysts. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1959, *82*, 853–861.
27. PARKER, D. Benign cysts of adrenal gland; case report. *J. Urol.*, 1952, *68*, 1–5.
28. PAUL, L. W., and JUHL, J. H. The Essentials of Roentgen Interpretation. Paul B. Hoeber, Inc., New York, 1959, pp. 316–317.
29. RACK, F. J., and EIBEN, R. M. Calcification of adrenal gland in infants and children. *J. Pediat.*, 1951, *39*, 618–622.
30. REIMANN, D. L., and GUYTON, W. L. Cysts of adrenal gland with case report. *Am. J. Path.*, 1947, *23*, 479–483.
31. SAMUEL, E. Calcification in suprarenal neoplasms. *Brit. J. Radiol.*, 1948, *21*, 139–142.

32. SCHWARTZ, O., and FINK, H. Calcification and ossification of adrenals in nine-month old infant. *J. Pediat.*, 1956, *48*, 334–340.

33. SNELLING, C. E., and ERB, I. H. Hemorrhage and subsequent calcification of suprarenal. *J. Pediat.*, 1935, *6*, 22–41.

34. STEVENS, R. C., and TOMSYKOSKI, A. J. Bilateral adrenal hemorrhage and calcification. *Am. J. Dis. Childhood*, 1954, *87*, 475–477.

35. THOMPSON, T. Addison's disease: value of radiography in direct diagnosis. *Lancet*, 1930, *2*, 785–787.

36. THORSTAD, M. J. Adrenal apoplexy. *Am. J. Surg.*, 1942, *55*, 44–48.

37. WEENS, H. S., and GOLDEN, A. Adrenal cortical insufficiency in infants simulating high intestinal obstruction. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1955, *74*, 213–219.

38. WIDMANN, B. P., OSTRUM, H. W., and FREED, H. Practical aspects of calcification and ossification in various body tissues. *Radiology*, 1938, *30*, 598–609.

39. ZUCKNER, J. Adrenal cysts. *Arch. Path.*, 1950, *50*, 468–474.

Vol. 86, No. 3

VESICOURETERAL REFLUX IN CHILDREN*

By DONALD R. ROONEY, M.D.†
MARIETTA, GEORGIA

THE purpose of this report is threefold: (1) to re-emphasize that the reflux of urine from the bladder into the ureter is an abnormal occurrence in children or in patients of any age; (2) to outline a simple reliable method for roentgenographic demonstration of reflux; and (3) to briefly review the growing body of literature dealing with vesicoureteral reflux in children.

IS VESICOURETERAL REFLUX ABNORMAL IN CHILDREN?

The varied answers to this question from many physicians, including radiologists, and the paucity of information dealing specifically with reflux in the recent radiologic literature served as stimuli for this report.

Studies in Normal Children. If one were to design a scientific study in an attempt to answer this question, one might start by obtaining cystograms of 100 urologically asymptomatic children with negative urinalyses. Just such a study was performed by Jones and Headstream.[14] They obtained cystograms in 100 children who met the above criteria and found reflux in only 1 child. This child was subsequently found to have a trabeculated bladder with 180 cc. of residual urine due to bladder neck contracture. It is also interesting to note that none of the 100 children developed evidence of urinary infection following cystography. Other investigators have made similar observations prior to and after the above study. Kjellberg *et al.*[15] obtained cystograms of 101 children aged two days to thirteen years who were apparently free of urinary abnormalities and none were found to have vesicoureteral reflux. Keuhnelian examined 26 children who were judged to have normal vesicoureteral junctions and in none of these was reflux observed.[18] Iannaccone and Panzironi[13] studied 50 children aged ten days to six months with no urinary signs or symptoms and found transient reflux in 1, but they were unable to reproduce reflux in the same patient ten days or three and one half months later. Since then, they have been quoted by McGovern *et al.*[18] as not having observed reflux in children without actual or subsequent infection or progressive dilatation. Bunge[2] obtained cystograms of 24 normal children and adults aged nine months to seventy-four years and was unable to demonstrate reflux in these patients. Politano[21] examined over 50 normal children without noting reflux. Campbell,[3] in discussing the cystographic findings in 722 children, states: "Reflux was not observed in any unanesthetized child with a normal bladder."

Earlier reports by Kretschmer[16] and Gibson[9] have given rise to controversy concerning the abnormality of vesicoureteral reflux in children. Kretschmer found reflux in 4 of 11 children, but 1 had cystitis and pyelitis. It is suggested by others[18] that the remaining cases of reflux could have been caused by the irritating qualities of the instilled solutions, resulting in edema about the ureterovesical junction. Gibson demonstrated reflux in 2 of 43 apparently normal children. In this same series 2 other children were found to have vesical diverticula. This would seem to cast some doubt on how many of the 43 apparently normal children actually did have normal urinary tracts.

Thus, the presently available evidence indicates that cystograms rarely, if ever, demonstrate reflux in normal unanesthetized children without other evidence of urinary disease.

Studies in Children with Urinary Dis-

* From the Department of Radiology, Kennestone Hospital, Marietta, Georgia.
† Associate Radiologist, Kennestone Hospital and Instructor in Radiology, Emory University School of Medicine, Atlanta, Georgia.

orders. In contrast to the absence, or extreme rarity, of reflux in normal children is the fact that approximately 12 per cent or more children with urinary disorders have reflux demonstrated on cystograms. St. Martin *et al.*[23] observed reflux in 13.5 per cent of 74 children with urologic signs or symptoms. Forsythe and Whelan[7] found reflux in 13.7 per cent of 445 children with urinary symptoms despite the fact that the complaint was enuresis in 430 of these. Campbell[3] demonstrated reflux in 12 per cent of 722 children with urinary disease. Five hundred symptomatic children twelve years of age or younger were examined by Pasquier *et al.*,[20] and in 13.8 per cent reflux was found. Thirty-five per cent of 152 consecutive symptomatic children examined by McGovern *et al.*[18] had reflux. There were 290 consecutive children with urinary tract infection, but with no evidence of obstruction or neurologic disorder reported by Kjellberg *et al.*[15] and reflux was seen in 34 per cent. Reflux was demonstrated in 34 per cent of our last 50 consecutive children with urinary disorders.

Of even greater significance than the increased incidence of reflux in children with urinary complaints is the observation that when reflux is found there is almost always a demonstrable coexisting or causative urinary disorder. This observation is supported by our studies and by those of others. McGovern *et al.*[18] state that, in their series of 88 children demonstrating reflux, "100% have significant urinary abnormality besides reflux." In Campbell's[3] aforementioned study of the cystograms of 722 children, he did not observe reflux except when other demonstrable uropathy coexisted. In discussing his series of 500 cystograms of children, Pasquier *et al.*[20] state, "In every instance we have been able to demonstrate an adequate cause for the reflux."

PATHOPHYSIOLOGY OF REFLUX

The ureterovesical junction is anatomically constructed to prevent reflux. According to Campbell,[3] "Normally the ureter enters the bladder obliquely for a distance of 5.0 mm. in an infant and 20.0 to 25.0 mm. in an adult, thus forming an oblique valve arrangement with a thin overhanging ureteral meatal lip to prevent backflow of urine." Gruber[10] observed that the percentage of reflux varied inversely with the thickness of the bladder and with the length of the intravesical portion of the ureter. Melick and Naryka[19] have shown with the strain gauge, a highly accurate instrument for measuring intravesical and intraureteral pressures, that, as the bladder is distended with fluid, the pressure in the normal ureter rises and remains higher than the intravesical pressure. From their studies they believe that this is the normal mechanism by which reflux is prevented. They also observed that reflux occurs when the ureteral peristaltic pressure fails to rise sufficiently as the bladder pressure rises. This increase in ureteral pressure may be due in part to the bladder compressing the intramural ureter plus the added factor of ureteral peristaltic pressure. Edwards[6] and Hanafee and Turner[11] observed cineroentgenographically that children with bladder neck obstructions had reflux into atonic ureters. In cases with simple megaureter, active forward and reverse peristalsis was observed. Pasquier *et al.*,[20] in preliminary studies with combined cystograms and cystometrograms, have shown that there is reflux with low bladder volumes and pressures in some children, while in others large volumes and high pressures are necessary before reflux occurs. Briefly stated, there is some type of unidirectional valve mechanism in the intramural portion of the normal ureter. This valve action may be active or passive or both.

Infection. There are many disease states which are able to alter the function of the ureterovesical junction, thereby causing reflux. In the acute phase, infection may temporarily convert the intramural ureter into a semi-rigid tube by surrounding edema and induration. Auer and Seager[1] produced experimental edema at the ureterovesical junction by infiltrating fluid in this area

and were thereby able to produce reflux. Such changes are reversible and reflux may disappear as the infection and edema regress. This may be why the 12 per cent of symptomatic children in whom reflux went undiagnosed in past years have not all had persistent reflux leading to progressive renal damage and death. The unrecognized reflux presumably cleared as the infection subsided with or without therapy. A chronic inflammatory process at the ureterovesical junction may cause fibrosis and scarring, thus changing the distal ureter into a permanently noncollapsible, stiffened tube. Chronic infection might also convert the intramural ureter into an extramural structure, as suggested by Hutch,[12] thereby destroying the valve mechanism which normally would prevent reflux.

Obstruction. Another large group of urinary disorders associated with reflux includes lesions causing obstruction to the outflow of urine such as: posterior urethral valves, median bar, urethral stricture, and other related lesions (Fig. 1, *A*, *B* and *C*). Of 133 children with congenital bladder neck obstruction reported by Uson *et al.*,[26] 67 per cent had reflux. In this group with intravesical obstruction, the changes at the ureterovesical junction are secondary to the obstruction lesion. This group is quite susceptible to urinary infection due to stasis, which probably plays a large part in causing the reflux and vice versa. Eighty-one per cent in the series of Pasquier *et al.*[20] with reflux had infection in addition to bladder neck and intravesical obstruction.

Anomalies. Congenital anomalies such as reduplication of the kidney and ureter or ectopic ureteral implantation are responsible for many cases of reflux (Fig. 2, *A–C*; and 3, *A–E*). Other anomalies in the distal ureter or trigone have been reported.

Neurogenic Disorders. These may produce reflux in some patients (Fig. 4). Swenson *et al.*[25] state: "There is evidence that the parasympathetic system helps to mediate a reflex contraction of the lower ureter during emptying of the bladder. . . . The bladder in some patients with megaloureters is abnormal in function because of a diminution in parasympathetic ganglion cells. The megaloureters are a result of this bladder dysfunction. . . .Certainly most patients with megaloureters have free reflux from the bladder." Perhaps in some cases of megaureter there may be secondary loss of ganglia due to dilatation and atrophy. Other neurologic disorders such as myelomeningocele, faulty innervation of the ureter or bladder, acquired paraplegia, or other types of "neurogenic bladder" may be responsible for the reflux. Only 3 per cent of the patients of Pasquier *et al.*[20] were thought to have neurogenic dysfunction as the cause of the reflux.

The etiology of vesicoureteral reflux is quite varied. Although infection, obstruction or anomalies account for most cases, combinations of these and other causes may interact in the same patient to produce reflux.

DIAGNOSIS

The presenting problem in almost all of our cases of children with reflux was unexplained recurring episodes of pyuria and fever. Flank pain during urination has been mentioned by Campbell[3] as a symptom associated with reflux. Apparently reflux occurs in these children as intravesical pressure rises during micturition. The resulting distention of the upper urinary tract by the refluxed urine produces the flank pain. The ability of a child to pass appreciable volumes of urine when he is allowed to empty his bladder two or three times in sucession suggests the presence of reflux. In these patients, varying amounts of the refluxed urine are passed back into the bladder after it is initially emptied (Fig. 5*E*). Other signs and symptoms are nonspecific for reflux and include almost any urinary complaint.

St. Martin *et al.*[23] have shown that 78.5 per cent of their cases with serious reflux had normal pyelograms. We have made similar observations. Kjellberg *et al.*[15] and

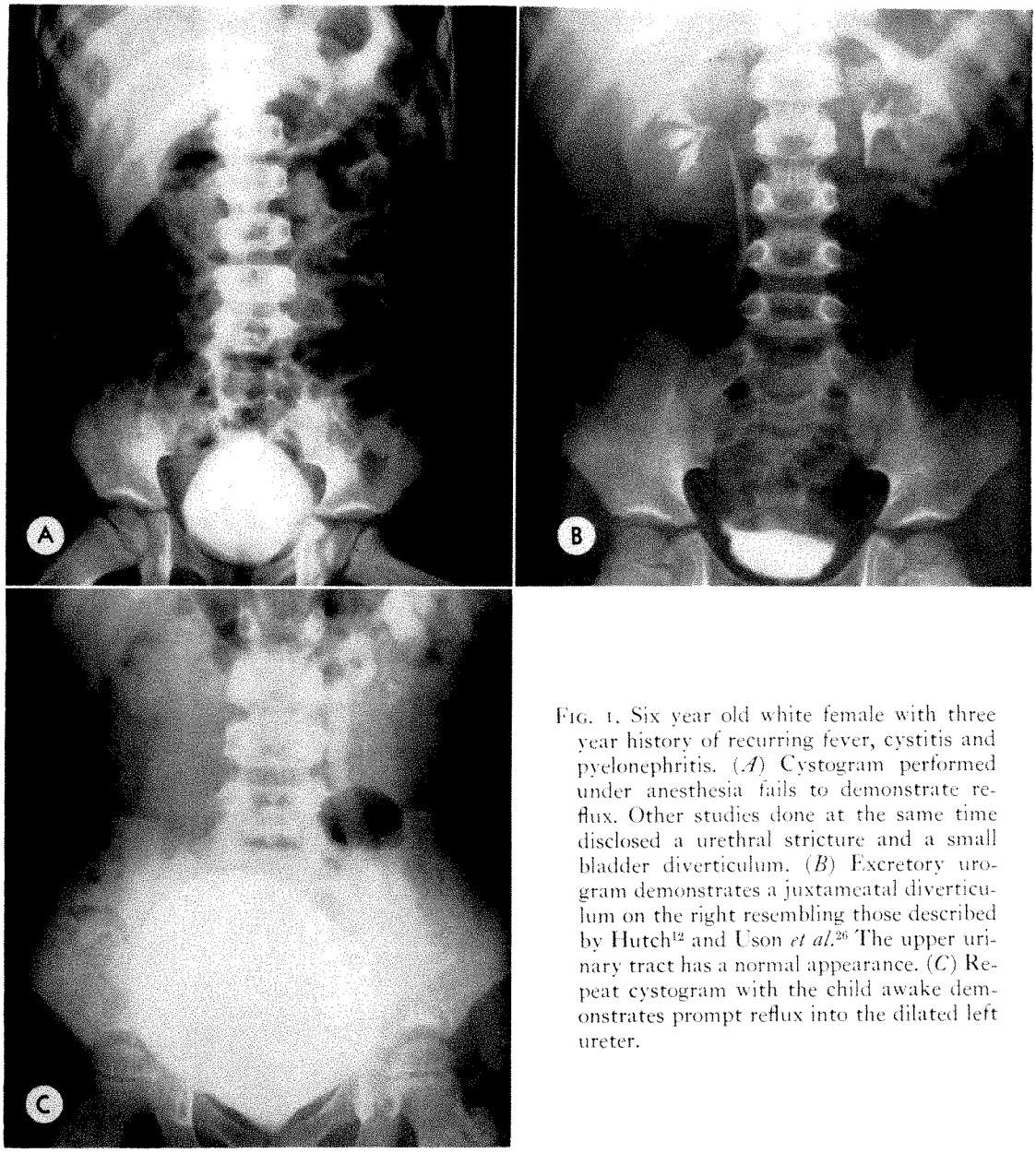


Fig. 1. Six year old white female with three year history of recurring fever, cystitis and pyelonephritis. (*A*) Cystogram performed under anesthesia fails to demonstrate reflux. Other studies done at the same time disclosed a urethral stricture and a small bladder diverticulum. (*B*) Excretory urogram demonstrates a juxtameatal diverticulum on the right resembling those described by Hutch[12] and Uson *et al.*[26] The upper urinary tract has a normal appearance. (*C*) Repeat cystogram with the child awake demonstrates prompt reflux into the dilated left ureter.

Stewart[24] report that reflux cannot be predicted from the cystoscopic appearance of the ureteral oriffice. Thus, we see that diagnostic studies in children with urinary problems are incomplete without a test for reflux (Fig. 5, *A–F*). Only two types of tests are available at present.

One method consists of filling the bladder with a solution containing a small tracer dose of radioactive iodine and then surveying the kidney areas for radioactivity with two continually recording devices. This method as presented by Winter[28] has certain advantages, but the expense and the scarcity of the necessary equipment limit its widespread use.

The second method, cystography, is simple and can be easily performed in any radiology department. There have been a few reports[2,4,5,8,24] dealing with variations

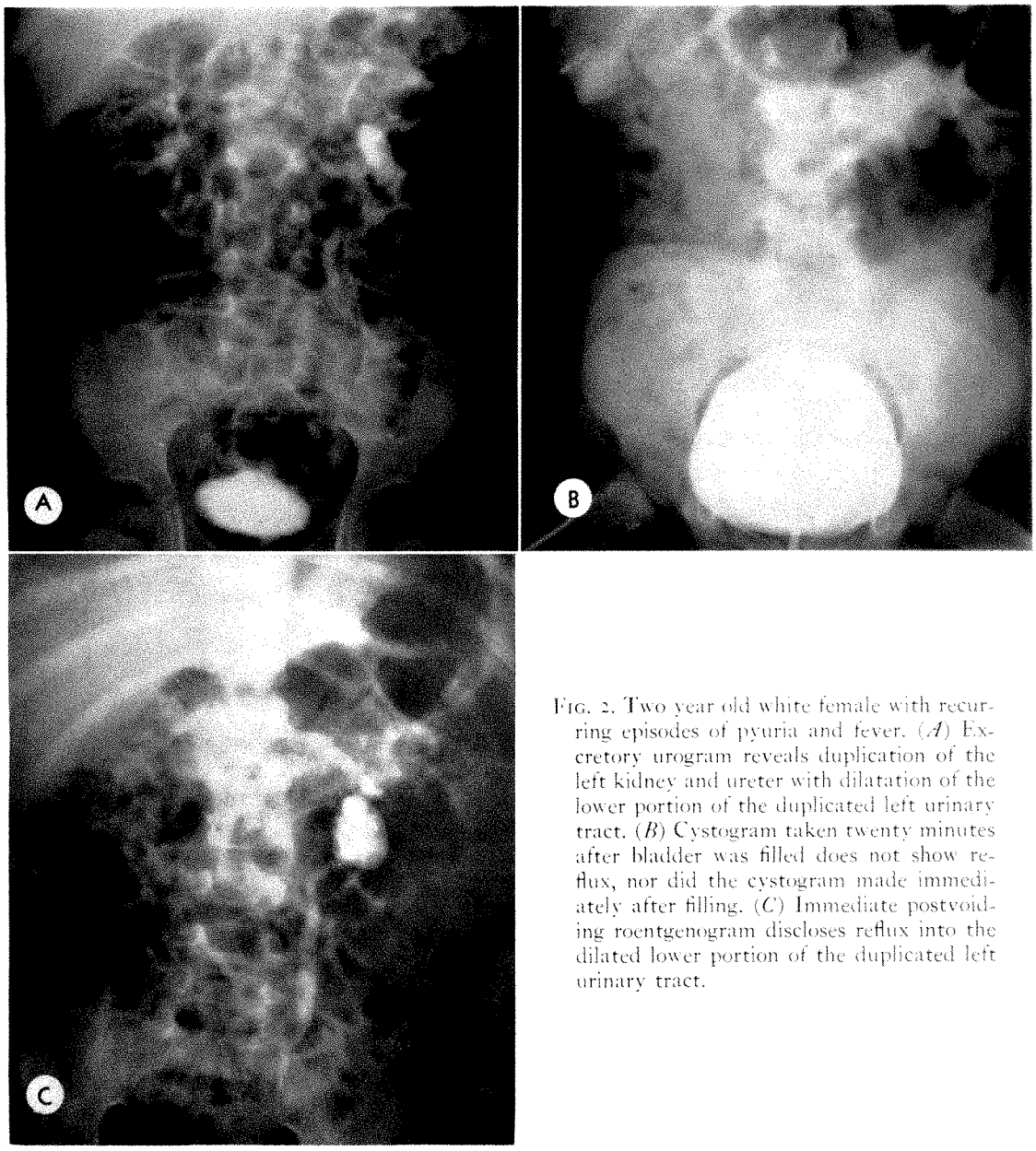


FIG. 2. Two year old white female with recurring episodes of pyuria and fever. (*A*) Excretory urogram reveals duplication of the left kidney and ureter with dilatation of the lower portion of the duplicated left urinary tract. (*B*) Cystogram taken twenty minutes after bladder was filled does not show reflux, nor did the cystogram made immediately after filling. (*C*) Immediate postvoiding roentgenogram discloses reflux into the dilated lower portion of the duplicated left urinary tract.

and refinements in the techniques for best demonstrating reflux. These have definite advantages but the equipment and time required for some of them might tend to discourage their adoption as routine diagnostic procedures. It would seem desirable to emphasize the need for a simple test on all pediatric urologic cases rather than to stress an elaborate one which would only be used on a few children.

PROCEDURE

Certain pitfalls must be avoided in order to prevent false negative or false positive findings of reflux in cystograms of children.

1. The child should not be anesthetized. We have observed 3 children who showed no reflux when cystography was done under general anesthesia but who were found to have marked reflux when these

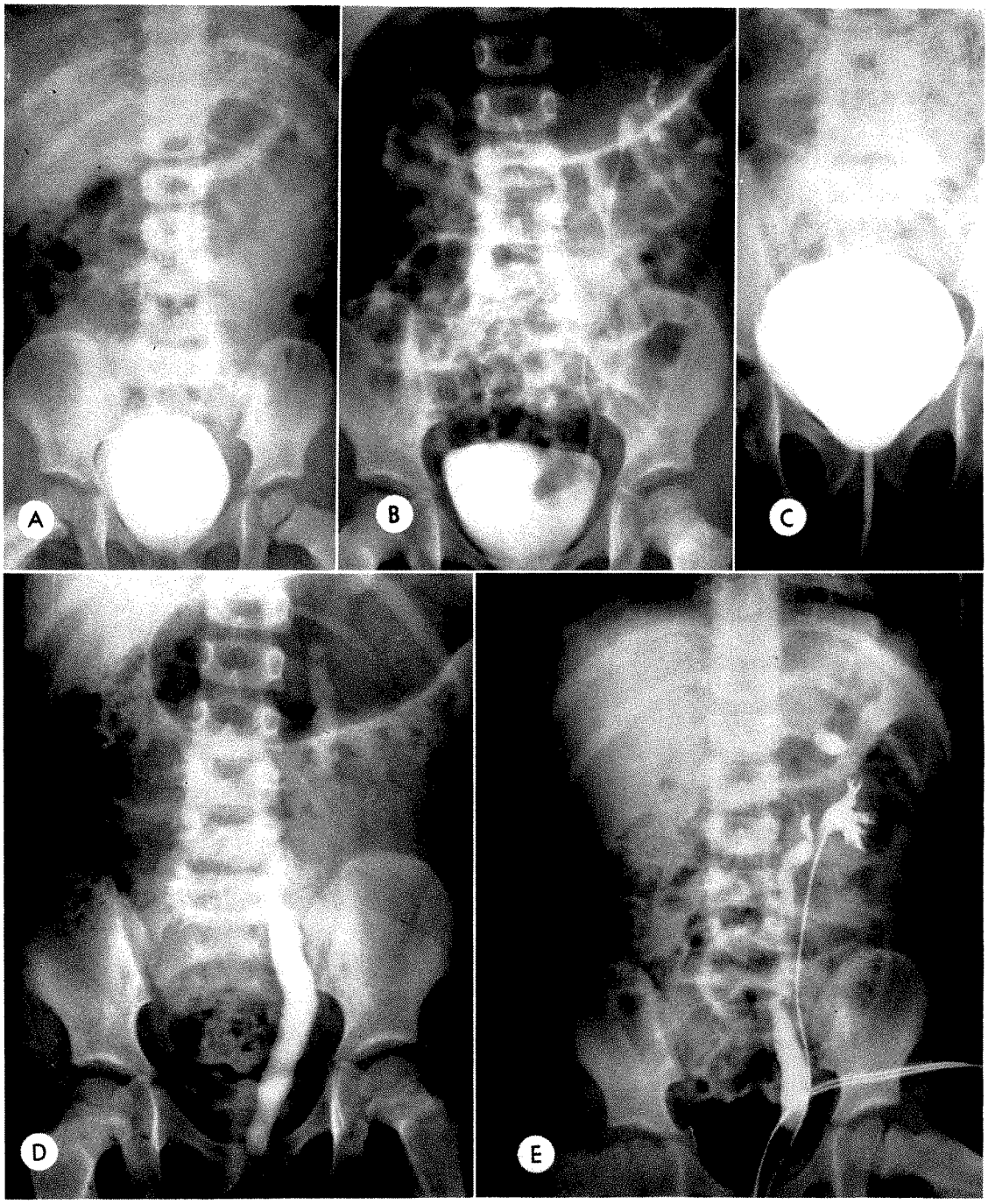


FIG. 3. Three year old white female with fifteen months' history of recurring pyuria and fever. (*A*) Cystogram taken with patient under general anesthesia fails to demonstrate reflux. (*B*) Excretory urogram fails to depict any dilatation or other intrinsic abnormality of the opacified structures. The left kidney is slightly lower than the right, but no opaque medium is seen above it to confirm the presence of a double left kidney. (*C*) Cystogram taken with patient awake. No reflux was seen on immediate or delayed roentgenograms with the bladder filled. (*D*) Immediate postvoiding roentgenogram demonstrates reflux into a dilated ureter from the upper portion of the duplicated left kidney. Note the low midline termination of this ureter. (*E*) On repeat retrograde study, both portions of the duplicated left tract are now depicted. An ectopic ureteral orifice from the dilated ureter was located in the urethra, which explains why this structure was opacified only on the postvoiding cystogram.

studies were repeated with the child awake (Fig. 1, *A–C;* and 3, *A–E*). Conversely, ureteral regurgitation may be seen in an anesthetized child with a normal bladder who demonstrates no reflux on repeat cystograms made while he is awake.

2. A cystogram should be taken immediately after filling the bladder. In some of our patients this was the only roentgenogram which demonstrated reflux.

3. A delayed cystogram should be taken while the bladder remains distended. Most children with reflux will demonstrate it during the first twenty minutes. To lengthen this part of the examination one or two hours or more, as some suggest, might discourage the widespread use of cystography.

4. A voiding or immediate postvoiding cystogram must be taken. In actual practice the voiding cystogram is not always feasible, since most of the patients are too young to cooperate fully. In these cases we have effectively used an immediate postvoiding cystogram. We commonly see reflux only on this one film (Fig. 2 *C;* and 3 *D*). This film also permits visualization of reflux into the distal ureter which might otherwise by obscured by the opacified bladder. St. Martin *et al.*[23] were able to increase their diagnostic accuracy of reflux 50 per cent by utilizing the postvoiding cystogram. If there is any delay in obtaining a postvoiding cystogram, a false negative finding will result, because the ureter will have emptied the refluxed opaque medium back into the bladder (Fig. 5*E*) and this will be misinterpreted as residual urine.

5. Always perform cystography first so that opaque media from excretory or retrograde pyelography will not obscure or be confused with reflux.

6. Use a meticulously aseptic technique when performing the catheterization.

7. Do not use irritating solutions, since the resulting edema may cause reflux.

8. Do not overdistend the bladder. Allow the bladder to drain completely through the catheter before instilling the opaque medium.

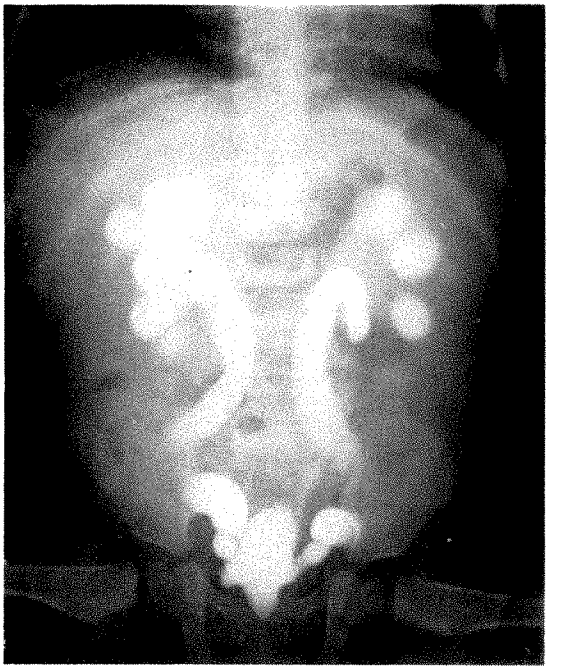


FIG. 4. Three year old white female who had had repeated urinary infections since the age of seven months. She also had partial paralysis of the lower extremities and clubbed feet and was later found to have an extradural lipoma in the lumbosacral canal. This cystogram reveals a deformed, contracted bladder and prompt bilateral reflux into a grossly dilated upper urinary tract.

9. Obtain repeat cystograms on children with persistent unexplained urinary infection if reflux is not demonstrated on the first examination, since, in some patients reflux is an elusive phenomenon.

10. Careful attention should be paid to roentgenographic technique in order to minimize motion and other factors which might obscure minimal reflux. Always obtain a preliminary roentgenogram so that technical factors may be corrected if necessary. Be certain that the kidney and bladder regions are completely included on the film.

TECHNIQUE

Utilizing our errors, made in the past, suggestions in the literature, and the experience gained from our last 85 cystographic examinations, the following technique has been gradually developed for obtaining routine cystograms in children. A routine cystogram series is illustrated in Figure 5, *A–D.*

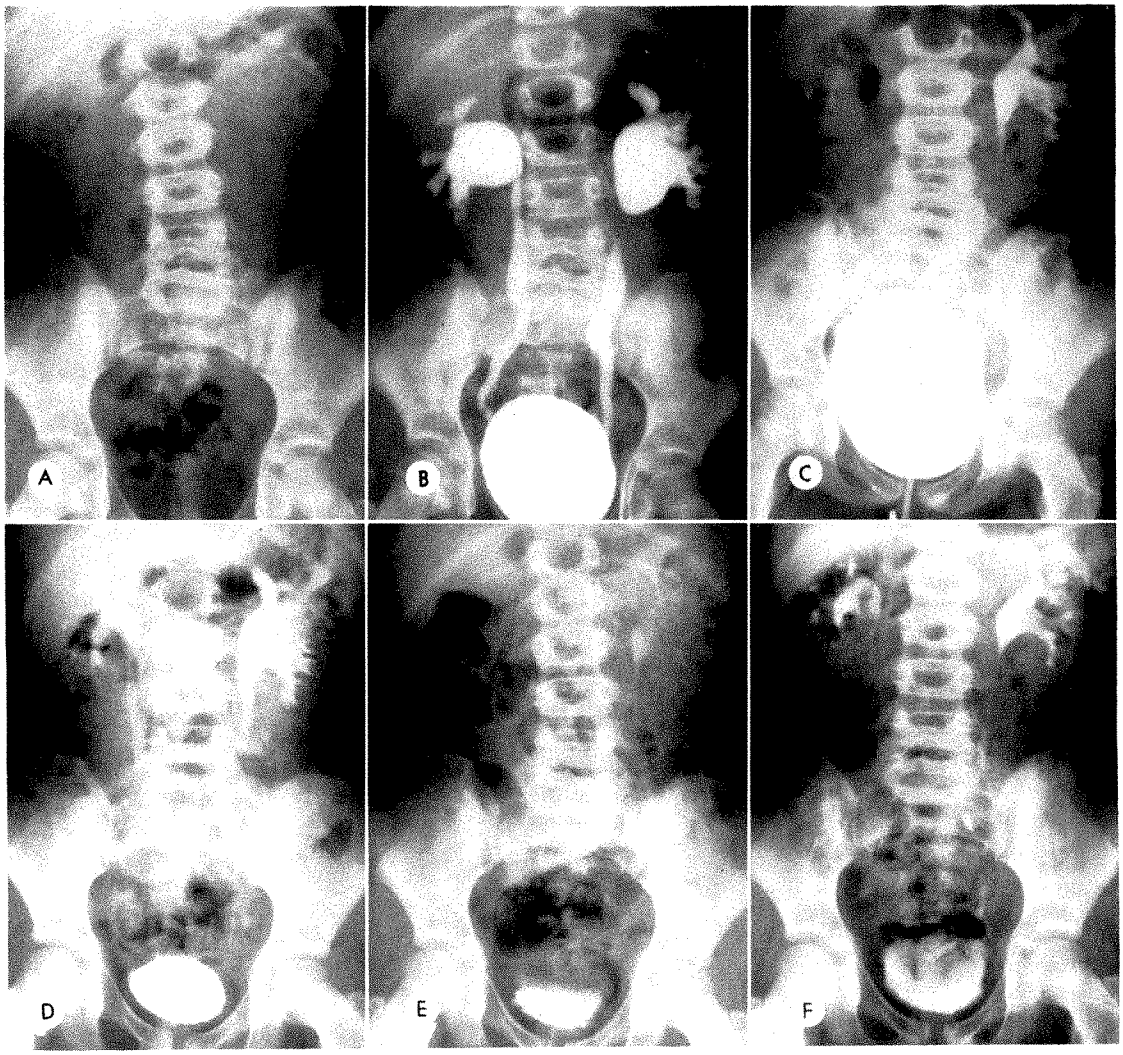


FIG. 5. Four year old white female with a two year history of recurring episodes of fever and pyuria. Subsequently, at the time of bladder neck revision, stenosis of the vesical neck was observed. Figures *A* through *D* constitute the routine cystograms. (*A*) Preliminary scout roentgenogram. (*B*) Cystogram taken immediately after the bladder is filled shows prompt bilateral reflux into slightly dilated ureters, pelves and calices. (*C*) On twenty minute delayed cystogram, some of the refluxed medium is seen to have emptied back into the bladder. (*D*) Immediate postvoiding cystogram reveals that bilateral reflux has again occurred. Residual opaque medium remains in the bladder. (*E*) A second postvoiding cystogram discloses that all the refluxed medium on the right and most of it on the left has emptied into the bladder. Pseudo-residual opaque medium is still present in the bladder. This demonstrates two points: double and triple voiding techniques, as suggested for conservative treatment of reflux, are effective methods to help eliminate stasis; and delay in obtaining the postvoiding cystogram may result in failure to demonstrate reflux. (*F*) Excretory urogram taken immediately after the cystogram appears to be normal.

1. A survey roentgenogram of the abdomen is made.

2. Using aseptic technique, a small catheter is inserted into the bladder and taped in place. The bladder is emptied and this urine may be saved for cultures of other studies. A large piston or bulb syringe is then connected to the catheter with a suitable adaptor.

3. Dilute hypaque (30 cc. ampule diluted with sterile water to 120 or 150 cc.) is allowed to flow by gravity from the syringe

which is elevated above the patient to approximately 20 cm. Usually about 75 cc. of fluid is used in less than three minutes. After this, approximately 50 cc. more is injected under slightly increased pressure. If any of the contrast medium leaks around the catheter or if the child complains of discomfort, the injection is stopped and the catheter is clamped. A total of 100 to 150 cc. of the contrast medium is used in the average young child.

4. As soon as the injection is completed, the second exposure is made. This film is processed and inspected. If bilateral reflux has occurred, omit step five.

5. After twenty minutes the third film is exposed.

6. With the film and roentgen-ray tube in place for a fourth exposure, the catheter is withdrawn. This usually initiates micturition and the child is allowed to void into a pan on the roentgen-ray table. After urination is completed, the pan is quickly withdrawn and the fourth exposure is immediately made.

We now routinely adhere to the above method for obtaining cystograms of children. Since the technicians have become accustomed to this technique, the entire examination is usually completed in approximately twenty-five minutes and it entails only a few minutes of the radiologist's time to perform the catheterization and to inspect the films. Over 90 cystograms have been obtained in the past twelve months at Kennestone Hospital. Seventy-seven of these were on 50 children aged seven months to sixteen years. Reflux was demonstrated in 15, or 34 per cent, of the 44 children, 14 of whom were females. In 6 children the reflux was bilateral.

TREATMENT

It is not the purpose of this paper to fully discuss the management of children with vesicoureteral reflux. Emphasis has been placed on the significance of this abnormal finding and how to demonstrate it roentgenographically. Treatment is mentioned for the sake of completeness and to further stimulate the interest of radiologists in this problem.

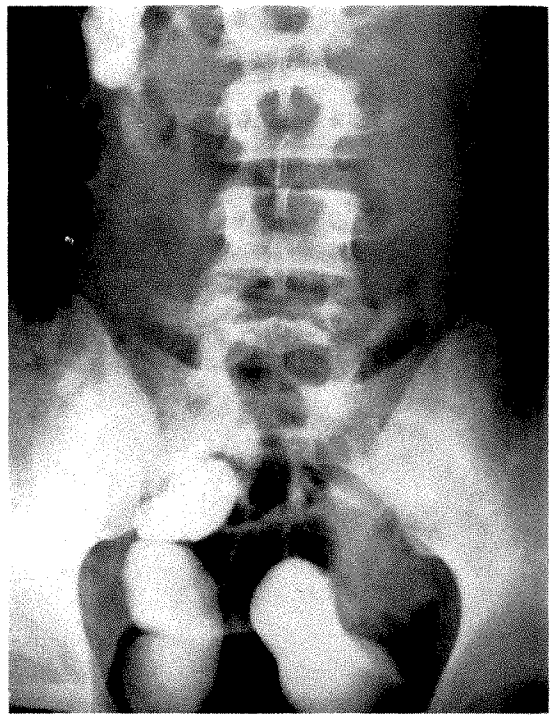


FIG. 6. White male, sixteen years of age, with uremia and advanced damage to ureters and kidneys, bilaterally. Persistent untreated reflux can produce this result. Every effort should be made to diagnose and treat vesicoureteral reflux promptly if changes such as these are to be avoided.

Briefly stated, the rationale of management is directed toward finding and eliminating not only the reflux, but the fundamental lesion causing reflux, first with conservative measures and then progressing to more radical methods in the persistent cases. Antibiotics or other drug therapy may be tried first. Measures designed to eliminate residual urine may be tried next, such as multiple voidings (Fig. 5*E*) or urethral dilatations. Leuzinger *et al.*[17] report that two-thirds of 190 children with reflux were greatly benefited following conservative therapy. If the problem persists, less conservative measures may be tried, such as transurethral resection, Y-V-plasty, or other surgical modifications of the bladder neck designed to eliminate obstruction. If reflux persists after correct-

ing the underlying abnormality, it is assumed that the changes at the ureterovesical junction are no longer reversible and surgical modification of the ureterovesical junction may then become necessary. Ureteral reimplantation with the creation of a submucosal tunnel, as described by Politano and Leadbetter,[22] has been quite successful in correcting reflux without producing ureteral obstruction. Politano[21] reports one failure and one partial failure in 26 children following this procedure. Vinson *et al.*[27] report that the Hutch type vesicoureteroplasty was 75 per cent successful in correcting reflux in 178 patients. Pasquier *et al.*[20] successfully corrected reflux with the Hutch type procedure in 10 of 11 children. McGovern, Marshall and Paquin,[18] in their excellent monograph dealing with vesicoureteral regurgitation, discussed 20 children who received vesicoureteroplasties and Y-V-plasties of the bladder neck. Twenty-eight refluxing ureters were reimplanted; reflux was seen postoperatively in only 1 patient and this ureter was subsequently reimplanted with no further reflux.

SUMMARY AND CONCLUSIONS

Vesicoureteral reflux rarely, if ever, is observed in the normal child. It is an abnormal occurrence in children, or in patients of any age, usually indicating an underlying urinary tract disorder. Persistent untreated reflux will lead to progressive and irreversible urinary tract damage. There are several causes for reflux. Urinary infection is important not only as a cause, but also as a result of reflux. Most cases are found to have an obstructing lesion at or below the bladder neck. Three fourths of the children with reflux may have normal pyelograms. Every child with an unexplained recurring urinary problem deserves a complete urologic evaluation which should include properly performed cystography. If the radiologist or urologist routinely obtains cystograms of these patients, his efforts will be rewarded with the demonstration of reflux in 12 per cent or more of them, and with the knowlege that many of these children will be spared the ravages of progressive renal damage (Fig. 6).

Therapy is directed toward eliminating the fundamental lesion and the reflux. Conservative therapy is tried first, and less conservative measures may be necessary in the persistent cases of reflux. Effective therapy is available for many children with vesicoureteral reflux; therefore, the radiologist should make every effort to demonstrate this abnormal finding.

Department of Radiology
Kennestone Hospital
Marietta, Georgia

The author wishes to express his thanks to Dr. Fred K. Schmidt for furnishing the clinical and operative findings in some of the illustrated cases and for his helpful criticism of the manuscript.

ADDENDUM

Since this manuscript was submitted, 91 cystograms have been obtained on 78 additional children. Reflux was demonstrated in 21 of these. Thus, in a two year period vesicoureteral reflux has been observed in 36 of the 128 symptomatic childrens' cystograms. In 13 of these 36 children, the reflux was bilateral. Surgical revisions have been performed on 26 of these 49 refluxing ureters which form the basis of a separate report which will appear shortly in the *Journal of the Medical Association of Georgia*.

REFERENCES

1. AUER, J., and SEAGER, L. D. Experimental local bladder edema causing urine reflux into ureter and kidney. *J. Exper. Med.*, 1937, *66*, 741–754.
2. BUNGE, R. G. Further observations with delayed cystograms. *J. Urol.*, 1954, *71*, 427–434.
3. CAMPBELL, M. F. Principles of Urology. W. B. Saunders Co., Philadelphia, 1957, p. 59 and 80.
4. CHRISTENSEN, E. R. Cystography with controlled filling pressure in children. *Acta radiol.*, 1959, *52*, 426–432.
5. DEAN, A. L., JR., LATTIMER, J. K., and MCCOY, C. B. Standardized Columbia University cystogram. *J. Urol.*, 1957, *78*, 662–668.
6. EDWARDS, D. Cine-radiology of congenital bladder-neck obstruction and megaureter. *Brit. J. Urol.*, 1957, *29*, 410–415.
7. FORSYTHE, W. I., and WHELAN, R. F. Occurrence and significance of vesico-ureteral reflux

in children. *Brit. J. Urol.*, 1958, *30*, 189–197.
8. Fulton, H. Delayed cystography in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *78*, 486–491.
9. Gibson, H. M. Ureteral reflux in normal child. *J. Urol.*, 1949, *62*, 40–43.
10. Gruber, C. M. Comparative study of intravesical ureters (uretero-vesical valves) in man and experimental animals. *J. Urol.*, 1929, *21*, 567–581. Also Uretero-vesical valve. *J. Urol.*, 1929, *22*, 275–292.
11. Hanafee, W., and Turner, R. Some uses of cine-fluorography in urologic diagnostic problems. *Radiology*, 1957, *73*, 733–739.
12. Hutch, J. A. Nonobstructive dilatation of upper urinary tract. *J. Urol.*, 1954, *71*, 412–420.
13. Iannaccone, G., and Panzironi, P. E. Ureteral reflux in normal infants. *Acta radiol.*, 1955, *44*, 451–456.
14. Jones, B. W., and Headstream, J. W. Vesicoureteral reflux in children. *J. Urol.*, 1958, *80*, 114–115.
15. Kjellberg, S. R., Ericsson, N. O., and Rudhe, U. The Lower Urinary Tract in Childhood: Some Correlated Clinical and Roentgenologic Observations. Year Book Publishers, Inc., Chicago, 1957, pp. 172–266.
16. Kretschmer, H. L. Cystography. Its value and limitations in surgery of bladder. *Surg., Gynec. & Obst.*, 1916, *23*, 709–717.
17. Leuzinger, D. E., Lattimer, J. K., and McCoy, C. B. Reflux is dangerous but not always disastrous: conservative treatment often effective. *J. Urol.*, 1959, *82*, 294–303.
18. McGovern, J. H., Marshall, V. F., and Paquin, A. J., Jr. Vesico-ureteral regurgitation in children. *J. Urol.*, 1960, *83*, 122–149.
19. Melick, W. F., and Naryka, J. J. Pressure studies of normal and abnormal ureter in children by means of strain gauge. *J. Urol.*, 1960, *83*, 267–276.
20. Pasquier, C. M., Jr., St. Martin, E. C., and Campbell, J. H. Further observations on problems of vesicoureteral reflux. *South. M. J.*, 1959, *52*, 967–974.
21. Politano, V. A. Vesicoureteral reflux in children. *J.A.M.A.*, 1960, *172*, 1252–1256.
22. Politano, V. A., and Leadbetter, W. F. Operative technique for correction of vesicoureteral reflux. *J. Urol.*, 1958, *79*, 932–941.
23. St. Martin, E. C., Campbell, J. H., and Pasquier, C. M. Cystography in children. *J. Urol.*, 1956, *75*, 151–159.
24. Stewart, C. M. Delayed cystography and voiding cystoureterography. *J. Urol.*, 1955, *74*, 749–759.
25. Swenson, O., MacMahon, H. E., Jaques, W. E., and Campbell, J. S. New concept of etiology of megaloureters. *New England J. Med.*, 1952, *246*, 41–46.
26. Uson, A. C., Johnson, D. W., Lattimer, J. K., and Melicow, M. M. Classification of urographic patterns in children with congenital bladder neck obstruction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *80*, 590–602.
27. Vinson, C. E., Hutch, J. A., and Bunts, R. C. Evaluation of vesicoureteral plasty for reflux. *J. Urol.*, 1957, *78*, 611–619.
28. Winter, C. C. New test for vesicoureteral reflux: external technique using radioisotopes. *J. Urol.*, 1959, *80*, 105–111.

INTRALUMINAL CALCIFICATION OF THE INFERIOR VENA CAVA*

By EDWARD B. SINGLETON, M.D., *and* HARVEY S. ROSENBERG, M.D.
HOUSTON, TEXAS

IN THE adult patient, the roentgenographic visualization of intra-abdominal calcification is a frequent finding. The calcification may exist as one or more phleboliths, atherosclerosis of the aorta or its branches, mesenteric lymph node calcification, or a variety of other forms which are readily recognized by roentgenographic means. Calcification within the abdomen of a child, however, is certainly more unusual and often implies a serious condition such as neuroblastoma, teratoma, or, less commonly, an embryoma or Wilms' tumor. Benign forms of calcification, although commonly seen on roentgenograms of the adult abdomen, are infrequent in the pediatric age group. Within the past year we have seen 2 children, who underwent roentgenologic examinations for entirely different reasons, in whom a localized area of intra-abdominal calcification was discovered which at laparotomy was found to lie within the lumen of the inferior vena cava and to be attached to its wall. We have been unable to find similar cases in our teaching file and in a review of the literature.

REPORT OF CASES

CASE 1. J.A.H. (88598) was a six week old white female who was admitted to the hospital because of continued vomiting and poor weight gain. The first hospital admission was at three weeks of age for similar complaints and a diagnosis of hypertrophic pyloric stenosis was made. Laparotomy was performed but the pyloric muscle appeared normal. Gastrointestinal examination had not been performed prior to this surgical procedure. The patient continued to have periodic vomiting and three days after the laparotomy developed diarrhea which was treated successfully with kanamycin and furazone. Prior to institution of chemotherapy, *Klebsiella pneumoniae* and *Shigella A* were cultured from the stool specimen. Because of continued periodic vomiting episodes, roentgenologic examinations of the gastrointestinal tract and urinary tract were performed. Both examinations showed an oval shaped area of calcific density located posterior to the stomach and extrinsic to the kidneys.

The family history was noncontributory and both parents were living and well. The mother's pregnancy was uncomplicated and the delivery and immediate postnatal period were in all respects normal. The patient had been on many different formulas in an attempt to correct the vomiting difficulties but none of these had been successful.

Physical examination showed a well developed, but slightly undernourished baby who was moderately dehydrated. A healed recent abdominal incision was identified, marking the recent laparotomy. There was no evidence of visible abdominal peristaltic waves, nor were there abnormal masses palpated through the abdominal wall. The lower extremities were normal without evidence of edema.

Laboratory studies showed a normal complete blood cell count and urinalysis, and blood chemistries, including sodium, potassium and carbon dioxide determinations, were within normal limits. The VDRL was nonreactive and examination of a stool specimen showed normal trypsin activity.

Roentgen examination of the upper gastrointestinal tract showed no abnormality of the esophagus, stomach or small intestine. However, on all of the roentgenograms there was an oval shaped area of calcific density which measured nearly 3 cm. in length. It was located anterior to the body of L-1 and posterior to the stomach (Fig. 1, *A* and *B*).

In order to investigate the calcification seen on the roentgenograms, an exploratory laparotomy was performed at which time a hard mass was identified within the lumen of the inferior

* From the Departments of Radiology and Pathology, Texas Children's Hospital, Houston, Texas. Presented at the Third Annual Meeting of the Society of Pediatric Radiology, Atlantic City, September 26, 1960.

vena cava. Venotomy was performed and the mass was found to be attached to the anterior wall of the vena cava just above the renal veins. The lesion was removed from its attachment and the venotomy repaired.

Grossly, the specimen was an oval yellow-brown structure with a coarsely granular surface measuring 1.7×0.9×0.3 cm. (Fig. 2*A*). Cut section disclosed an outer irregular calcific capsule with an encapsulated nodule of fibrous tissue. The central portion of this structure was made up of loosely arranged sheets of young stellate fibroblasts and a few thin-walled blood vessels. Hemosiderin laden macrophages were identified within the fibrous tissues. The calcification was present beneath the capsule and was surrounded by a thin compressed layer of mature fibroblasts (Fig. 2*B*). The pathologic impression was that of a calcified organized thrombus from the inferior vena cava.

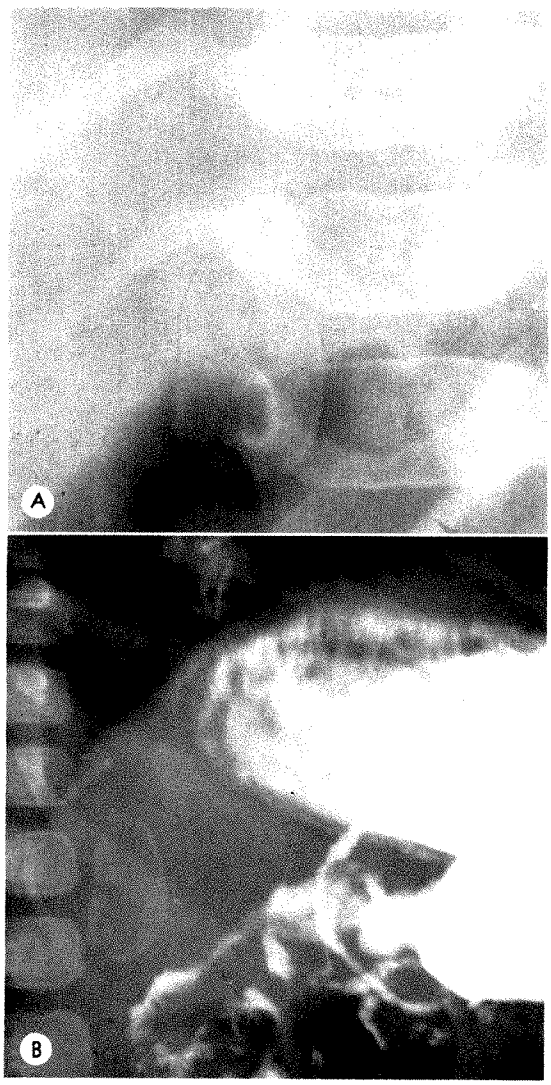


FIG. 1. Case I. (*A*) Anteroposterior roentgenogram of the right upper quadrant shows an ill-defined area of density overlying the right lateral border of L-1. (*B*) Lateral roentgenogram shows the oval shaped area of calcification to be posterior to the stomach and duodenum.

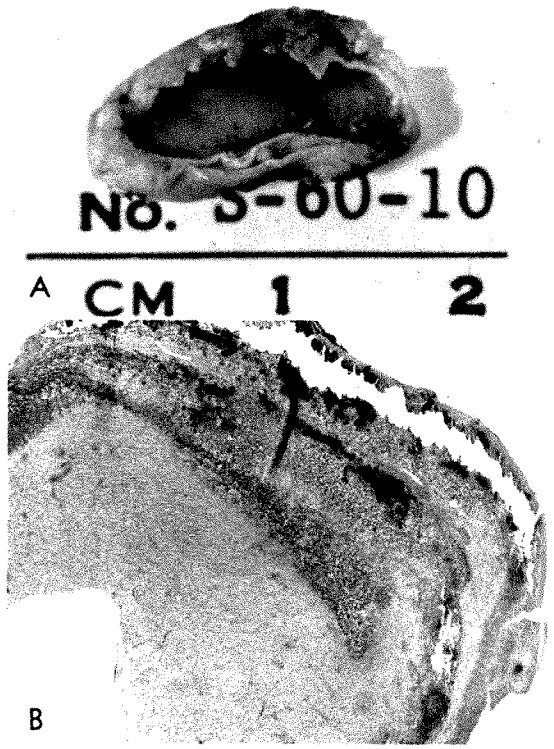


FIG. 2. Case I. (*A*) Gross specimen. (*B*) Histologic section showing features of a calcified organized thrombus.

The patient's recovery following this procedure was uneventful, and, after repeated instructions to the mother in the handling of her baby, the infant stopped vomiting and is no longer a feeding problem.

CASE II. P.A.L. (93111) was a four year old white female who was admitted to the hospital for investigation of abdominal pain which had been present intermittently for one month. The discomfort occurred usually in the evening and was located in the left flank and left lower quadrant. In addition, the patient had lost approximately five pounds of weight in the past four weeks. Two days prior to admission, the mother noted that the urine was dark brown in color and on the evening before admission the

patient awakened during the night complaining of severe abdominal pain and this was followed by gross hematuria.

The past history was completely normal other than the usual childhood diseases. Both parents have been in good health and there was no history of maternal illness during gestation.

The abnormal physical findings were limited to the abdomen. There was slight tenderness and spasm over the left flank and left costovertebral angle. A firm, slightly movable, nodular mass was palpated in the left upper quadrant.

Laboratory studies showed a hemoglobin of 12.2 gm. with a hematocrit of 36 mm. The white blood cell count was 10,700 with a normal differential. Urinalysis showed the urine to be dark red in color and loaded with red blood cells. Blood chemistries, consisting of blood urea nitrogen, calcium and phosphorus values, were within normal limits.

Roentgenograms of the abdomen showed a normal distribution of intestinal gas. The right renal shadow was of normal size, but the left kidney appeared enlarged. A triangular shaped area of calcific density having the configuration of a tooth and measuring 1.5 cm. in length was identified to the right of the twelfth dorsal interspace (Fig. 3, *A*, *B* and *C*). Excretory urograms showed nonfunction of the left kidney and retrograde studies showed a mass occupying and distorting the left renal pelvis. The superior and inferior pole calyces were markedly dilated, with the remaining portions of the kidney apparently occupied by a large renal neoplasm. Because of the tooth-like area of calcification, retroperitoneal teratoma with invasion of the kidney was suspected.

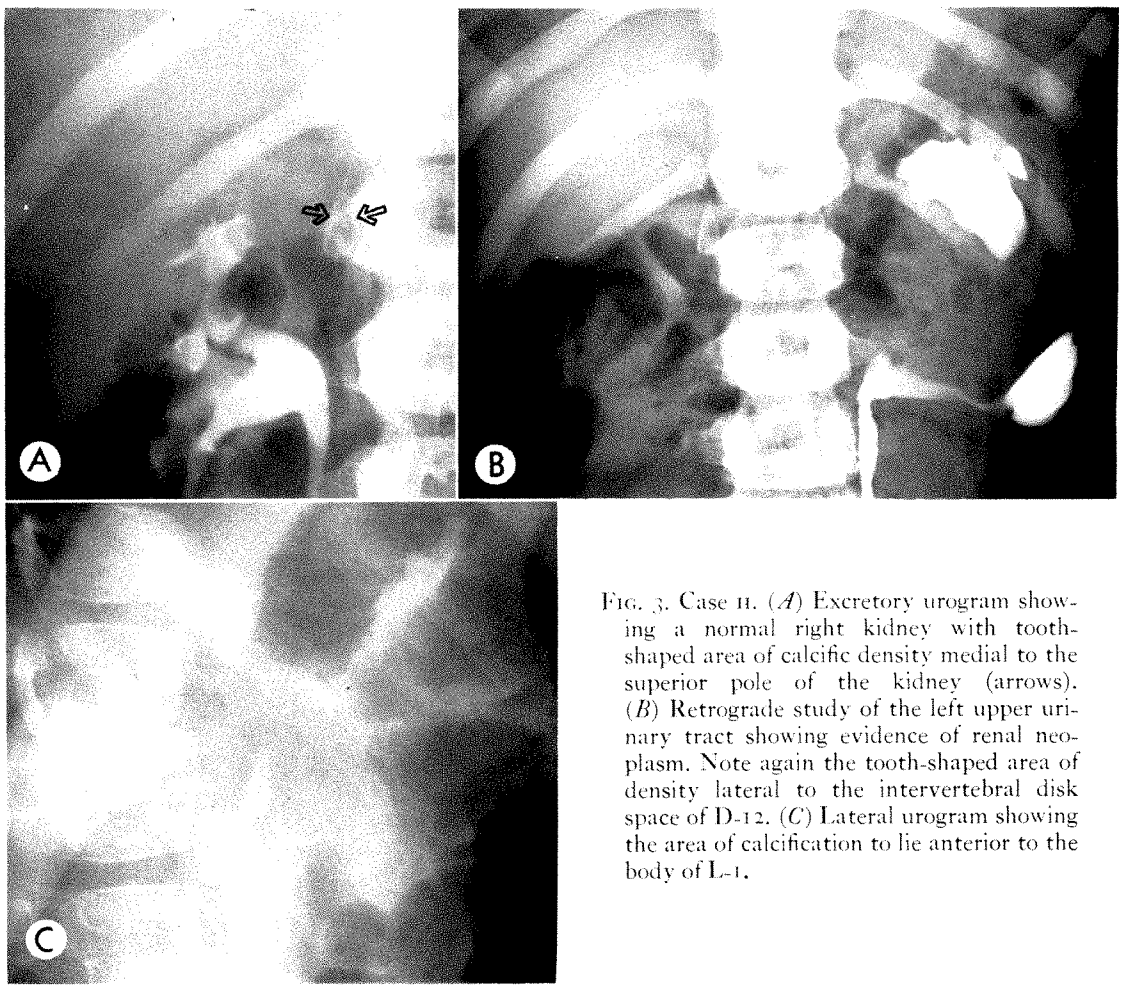


FIG. 3. Case II. (*A*) Excretory urogram showing a normal right kidney with tooth-shaped area of calcific density medial to the superior pole of the kidney (arrows). (*B*) Retrograde study of the left upper urinary tract showing evidence of renal neoplasm. Note again the tooth-shaped area of density lateral to the intervertebral disk space of D-12. (*C*) Lateral urogram showing the area of calcification to lie anterior to the body of L-1.

Surgical investigation showed a large neoplasm occupying the pelvis of the left kidney which had the gross appearance of a Wilms' tumor. Left nephrectomy was carried out and following this a small firm mass could be palpated in the inferior vena cava, at the area in which the calcific mass was identified on preoperative roentgenograms. Venotomy was performed and the calcific lesion was found to be attached by pedicle to the wall of the vena cava, just above the right renal vein. The lesion was removed without difficulty and the venotomy repaired. The patient made an uneventful recovery and the operative site was treated by external radiation. The patient has remained free of visible neoplasm to the present time, eight months following nephrectomy.

Histologic diagnosis of the renal lesion was Wilms' tumor with vascular and lymphatic invasion. The calcific lesion which was removed from the inferior vena cava was a roughly oval, yellow-brown calcific structure measuring 1.2 ×0.6×0.3 cm. (Fig. 4*A*). The lesion, which had been removed in several fragments, contained a brittle central core. Histologic examination showed the lesion to be an encapsulated amorphous structure which was densely calcified and focally ossified. There were broad areas of edematous connective tissues containing a few fibroblasts, but no evidence of active inflammation (Fig. 4*B*). The histologic diagnosis was organized thrombus of the inferior vena cava.

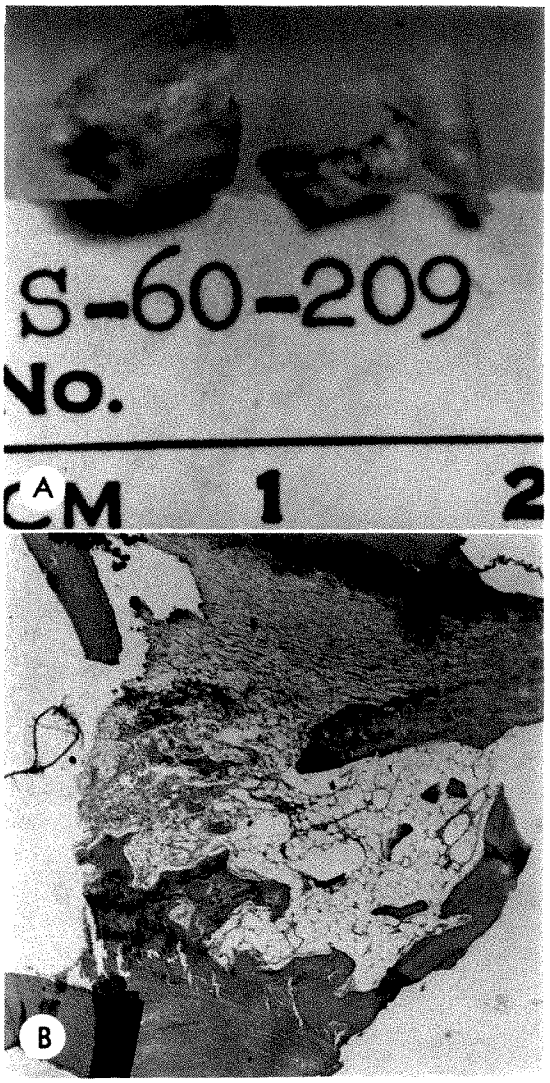


Fig. 4. Case II. (*A*) Gross specimen in fragments. (*B*) Histologic section showing bone formation within the organized thrombus.

DISCUSSION

There are a number of reports in the radiologic literature describing calcification of intra-abdominal veins, as well as calcified thrombi of these structures. This is especially prevalent in patients with Banti's syndrome[3,5,6] and in cases of endophlebitis involving the splenic vein, inferior mesenteric vein, gastroepiploic vein, superior mesenteric vein and portal vein. Li[4] credits Morgagni in 1769 and Baillie in 1793 with reporting calcification involving the inferior vena cava. However, there is no reason to believe that these cases were similar in origin or appearance to the 2 reported here. Although to our knowledge other cases of calcified thrombi affecting the inferior vena cava of children have not been reported, a few have been identified in other institutions.[2,7]

Experimentally, it is recognized that increased pressure within a vein, as well as phlebitis, may lead to thrombosis and this in time may undergo calcification and even later ossification. Neither of the patients described in this report had reason for increase in the pressure of the inferior vena cava such as cirrhosis, portal hypertension or cardiac decompensation. In each case the liver appeared grossly normal at the

time of laparotomy and the spleen was not enlarged. Neither was there a history of phlebitis involving systemic or visceral veins. It is conceivable that in the case of the patient with the suspected pyloric stenosis, the inferior vena cava was locally damaged with resulting thrombosis and calcification. However, the histologic appearance was not that of a recent thrombus but instead suggested that the process had been present for a longer period of time than would have been possible had the thrombosis developed at the time of the recent surgical procedure. In the second case it seems unlikely that the Wilms tumor would have resulted in calcification of the inferior vena cava. There was no evidence of extension of the neoplasm to the vena cava at this point.

Arey[1] has suggested that the thrombus may be secondary to severe dehydration, and, although the first case had a history of recurrent emeses, the infant was never considered to be severely dehydrated. In addition, the histologic appearance of the thrombus suggested that it antedated the period of dehydration. In the second case, there was no history of dehydration.

In addition, attempts to incriminate an embryonic defect have been unsuccessful. Consequently, we are unable to offer an exact etiologic explanation for the calcified vena caval thrombi exhibited by these 2 patients.

One can only postulate as to the eventual result of such a lesion. The possibility of extension of the thrombus with either eventual occlusion of the inferior vena cava or dislodgement of a portion of the thrombus with pulmonary infarction would appear inevitable. Consequently, the surgical removal of these lesions seems justified.

SUMMARY

Two children with calcified masses (probably thrombi) within the inferior vena cava are reported. The etiology is unknown but the possibility of a calcified thrombus of the inferior vena cava should be considered in the differential diagnosis of intra-abdominal calcifications.

Edward B. Singleton, M.D.
St. Luke's and Texas Children's Hospitals
Department of Roentgenology
6621 Fannin Street
Houston 25, Texas

REFERENCES

1. AREY, J. B. Personal communication.
2. BAKER, DAVID. Personal communication.
3. BLEICH, A. R., and KIPEN, C. S. Venous calcification in Banti's syndrome. *Radiology*, 1948, *50*, 657–660.
4. LI, P. L. Adaptation in veins to increased intravenous pressure, with special reference to portal system and inferior vena cava. *J. Path. & Bact.*, 1940, *50*, 121–136.
5. LINDORN, O. Chronic thrombosis of splenic and portal veins; clinical picture similar to Banti's disease. *Hygiea*, Stockholm, 1915, *77*, 705–738.
6. MAGOVERN, G. J., and MUEHSAM, G. E. Calcification of portal and splenic veins. AM. J. ROENTGENOL. & RAD. THERAPY, 1954, *71*, 84–88.
7. SILVERMAN, F. J. Personal communication.

VOL. 86, NO. 3

TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION TO THE PORTAL VENOUS SYSTEM: A CAUSE OF PULMONARY VENOUS OBSTRUCTION*

By RUSSELL V. LUCAS, JR., M.D.,† PAUL ADAMS, JR., M.D., RAY C. ANDERSON, M.D., RICHARD L. VARCO, M.D., JESSE E. EDWARDS, M.D., *and* RICHARD G. LESTER, M.D.
MINNEAPOLIS AND ST. PAUL, MINNESOTA

THE physician confronted with a cyanotic newborn infant must consider the possibility of total anomalous pulmonary venous connection to the portal venous system. This condition leads to death in early infancy. Since surgical correction is theoretically possible, action toward immediate diagnosis is mandatory if surgical salvage of these infants is to be accomplished.

Harris and associates[8] have stressed the value of the thoracic roentgenogram in suggesting the presence of this anomaly. When the thoracic roentgenogram reveals fine stippled reticular pulmonary markings and a normal sized heart, the differential diagnosis in a cyanotic neonate is narrowed to pulmonary parenchymal disease or pulmonary venous obstruction.

Among the several causes of pulmonary venous obstruction is the condition discussed in this paper—total anomalous pulmonary venous connection with a vein of the portal system. This condition may occur alone, or it may be associated with other major cardiac anomalies, with different diagnostic and therapeutic problems in either circumstance. This report will consider only cases that are not associated with other cardiac anomalies. A presentation of the angiocardiographic observations correlated with the pathologic findings in total anomalous pulmonary venous connection to the portal venous system is the major purpose. Angiocardiography was diagnostic in 3 of the 4 patients seen by the authors, and, in retrospect, it could have been the basis of the diagnosis in the fourth patient.

In addition, the clinical, electrocardiographic, and roentgenographic features of our 4 cases will be compared with available data on the 29 other cases we have found in the literature. Description of the attempts at surgical correction of the anomaly in 2 cases will also be given.

CLINICAL REVIEW

A summary of the pertinent data in our 4 cases along with those of 29 cases collected from the literature[1–6,8–11,13,14,16–19] is given in Table 1.

Death occurred in infancy in all cases. Male infants predominated in a ratio of about 3 to 1. A cardiac murmur was infrequently heard. Cyanosis and dyspnea were almost universal. The electrocardiogram revealed right axis deviation and hypertrophy of the right ventricle in every case in which electrocardiographic findings were available.

The literature indicates that roentgenographic studies may lead to a definitive diagnosis of this anomaly. Routine roentgenographic studies reveal the cardiac size to be normal in the large majority of patients. Abnormal pulmonary vascular markings characterized by diffuse, small, irregular densities and described as "reticular markings," "pulmonary edema," and markings "similar to those in neonatal atelectasis" were present in almost all patients

* From the Departments of Pediatrics, Surgery, Pathology, and Radiology, University of Minnesota, Minneapolis, Minnesota, and the Department of Pathology, The Charles T. Miller Hospital, St. Paul, Minnesota.
This study was supported in part by Research Grants #H-5694 and H-3580 of the National Heart Institute, United States Public Health Service.
† Postdoctoral Research Fellow, National Heart Institute, National Institute of Health, United States Public Health Service.

TABLE I

PERTINENT DATA IN 33 CASES OF TOTAL ANOMALOUS CONNECTION OF THE PULMONARY VEINS TO THE PORTAL VENOUS SYSTEM

Source	Sex	Age at Death	Cyanosis*	Dyspnea*	Swallowing Symptoms*	Cardiac Murmur*	RVH on ECG*	Normal Heart Size*	Reticular Lung Markings*	Angiocardiography*	Surgical Intervention*
Literature (29 cases)	22 M 6 F	2 da. to 3 mo.	22/23	16/17	0/0	5/19	7/7	16/19	17/19	1/29	0/29
Present Authors' Case I	F	4½ mo.	+	+	+	0	+	+‡	+	+	0
Case II	M	5 wk. (PO)	+	+	+	Gr. II† systolic	+	+	+	+	died after surgery
Case III	M	5 wk. (PO)	+	+	+	Gr. I systolic	+	+	+	+	died after surgery
Case IV	M	2 wk.	+	+	+	Gr. II systolic	+	+	+	+	0
Total 33	M 25 F 7	2 da. to 4½ mo.	26/27	20/21	4/4	8/23	11/11	20/21	21/23	5/33	2/33

RVH—Right ventricular hypertrophy; ECG—electrocardiogram; PO—postoperative.
* The first figure indicates those patients with the finding and the second the number in which this finding was considered.
† On the basis of six grades.
‡ Normal at three days of age; cardiomegaly at seven days.

of whom roentgenograms of the lungs were described. Johnson and associates[9] reported the results of venous angiocardiography in one patient in whom contrast medium appeared in the region of the porta hepatis nine seconds after injection. To our knowledge, the present report is the only other description in the literature of the angiocardiographic findings in total anomalous venous connection to the portal venous system.

The following roentgenographic and pathologic findings were observed in our 4 cases.

REPORT OF CASES

CASE I.*

Roentgenographic Findings. Thoracic roentgenograms were taken in January 1954, of this three day old white female infant during evaluation of cyanosis and dyspnea. Roentgenographic study (Fig. 1*A*) demonstrated that the heart was normal in size and shape. Diffuse fine linear streaks and fine mottling were present in both lungs. A roentgenogram taken four days later revealed cardiac enlargement; furthermore, the fine mottling in the pulmonary fields was accentuated in comparison with that shown at the first examination.

At ten days of age, the patient was subjected to venous angiocardiography. While the patient was in the left anterior oblique position, 4.5 cc. of 35 per cent iodopyracet (diodrast) was injected into the right greater saphenous vein at the ankle. Serial roentgenograms were made at the rate of two per second for five seconds. Review of these roentgenograms showed contrast medium entering the inferior vena cava and progressing to the right atrium and right ventricle and, simultaneously, contrast material filling the left atrium. The pulmonary artery was large and distinct, and it opacified simultaneously with the ascending aorta.

Throughout the remainder of the study the right-sided chambers and the pulmonary artery remained opacified. No opacification of the pulmonary veins or reopacification of the left atrium was observed. A positive diagnosis was not made. Since the patient's condition appeared stable, she was discharged from the hospital.

The patient was re-admitted at three and one-half months of age for re-evaluation of her intensified episodic cyanosis. Thoracic roent-

* Data on this case were included in a previous report by Gott and associates.[7]

genograms revealed moderate cardiomegaly and, as earlier, a fine, lacy, stippled appearance in both pulmonary fields (Fig. 1 *B*). A second angiocardiographic study was performed, 7 cc. of 35 per cent diodrast being injected into the left brachiocephalic vein. Films were exposed at the rate of two per second with the patient positioned in the left anterior oblique. The contrast medium outlined the left innominate vein, the superior vena cava, and the right atrium. Opacification of the large right atrium was followed by simultaneous opacification of the right ventricle and left atrium, then followed simultaneous dense opacification of the aorta which was small and of the pulmonary artery which was large. Again, recirculation of contrast material did not occur, nor did the left side of the heart reopacify. The right atrium, right ventricle, and pulmonary artery remained opacified during the entire study. The patient was discharged on the day of the angiocardiographic study without a definitive diagnosis. One month later, at the age of four and one-half months, the patient suddenly became unconscious and was dead on arrival at the University of Minnesota Hospitals.

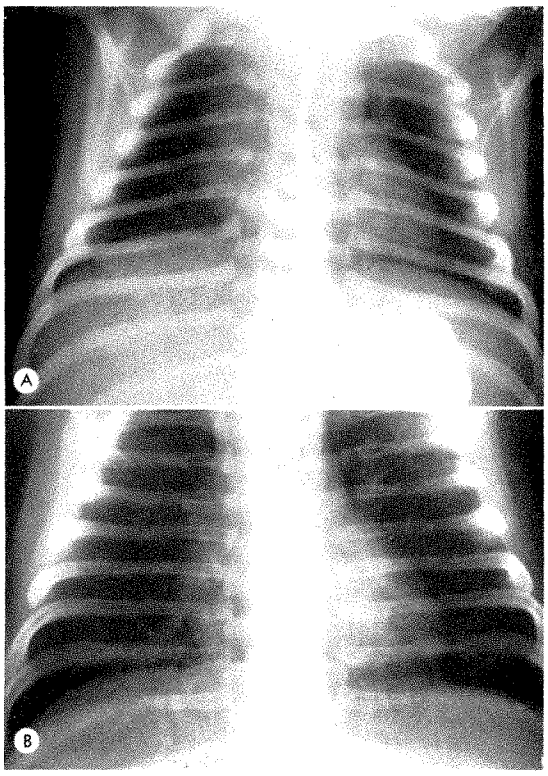


FIG. 1. Case 1. (*A*) Thoracic roentgenogram taken when patient was 3 days old shows normal-sized heart and reticular markings in pulmonary fields. (*B*) Thoracic roentgenogram at 3½ months shows no significant changes from earlier examination.



FIG. 2. Case 1. Frontal photograph of the thoracic and abdominal organs. The heart has been lifted up. The four pulmonary veins converge behind the left atrium to form a common pulmonary vein (C.P.V.). This common pulmonary vein is narrowed at its point of penetration through the esophageal hiatus of the diaphragm. It then becomes dilated and terminates (arrow) in the portal vein (P.) at the confluence of the superior mesenteric vein (M.) and the splenic vein (transected in this specimen). In the thorax the common pulmonary vein is in close association with the esophagus (E.). L.—liver; S.—stomach.

Pathologic Findings. Pertinent findings were restricted to the cardiovascular system. The lungs were expanded and appeared normal grossly. The aorta and pulmonary arteries arose in the normal manner and were normally interrelated. The ductus arteriosus was obliterated. When the heart was retracted upward (Fig. 2), the two pulmonary veins from each lung were observed to join near the midline to form a common trunk. This trunk descended inferiorly, immediately anterior to the esophagus, and it penetrated the diaphragm through the esopha-

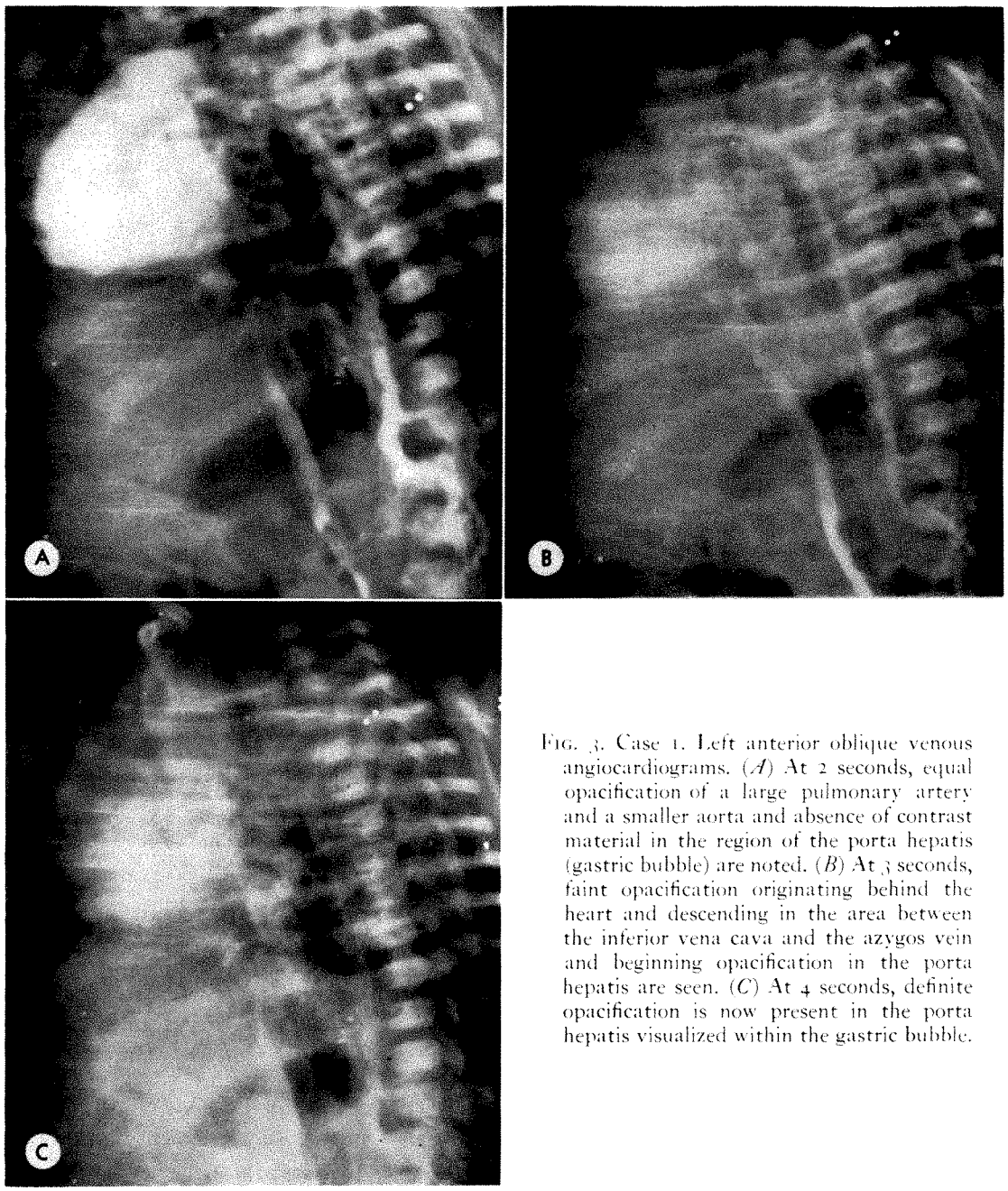


FIG. 3. Case 1. Left anterior oblique venous angiocardiograms. (*A*) At 2 seconds, equal opacification of a large pulmonary artery and a smaller aorta and absence of contrast material in the region of the porta hepatis (gastric bubble) are noted. (*B*) At 3 seconds, faint opacification originating behind the heart and descending in the area between the inferior vena cava and the azygos vein and beginning opacification in the porta hepatis are seen. (*C*) At 4 seconds, definite opacification is now present in the porta hepatis visualized within the gastric bubble.

geal hiatus along with the esophagus. This anomalous common pulmonary vein then joined the portal vein just above the confluence of the splenic and superior mesenteric veins. The anomalous common pulmonary venous trunk was 1 cm. in diameter at its termination at the portal vein. It had a narrow zone which lay in the thorax just above the diaphragm, where the diameter was 4 mm. No pulmonary veins joined the heart directly.

Internal examination of the heart revealed a valvular competent patent foramen ovale with the potential opening measuring 1×2 cm. The ventricular septum was intact, and the ventricular cavities as well as the semilunar and atrioventricular valves were of normal size. The left

atrium was small in relation to the right atrium.

It is noteworthy that upon retrospective review of the angiocardiograms obtained when the patient was three days old, we identified faint staining of a vascular channel extending from below the cardiac shadow to a point beneath the diaphragm. In addition, a spherical shadow of contrast medium was observed to lie in the area of the porta hepatis (Fig. 3, *A*, *B* and *C*). This opacification, which occurred late in the examination, had been overlooked during the initial interpretation of the roentgenograms. The shadows noticed retrospectively in this study are thought to represent the lower portion of the anomalous common pulmonary venous channel.

Case II.

Roentgenographic Findings. A four week old white male infant had thoracic roentgenography performed in July, 1959, during evaluation of cyanosis and dyspnea. The roentgenograms demonstrated normal cardiac size and contour. Both pulmonary fields showed a fine, diffuse stippling (Fig. 4). A diagnosis of pulmonary venous obstruction was made.

Three days later venous angiocardiography was performed. Six cubic centimeters of 90 per cent diatrizoic acid (hypaque-M) were injected into the right greater saphenous vein at the ankle while films were being exposed in frontal and lateral projections at the rate of five per second for six seconds. Initially, contrast material outlined the right and left atria. Later, simultaneous dense opacification of the large pulmonary artery and the small aorta occurred (Fig. 5, *A* and *B*). These vessels were in normal position. A patent ductus arteriosus was well visualized. On the late roentgenograms (Fig. 5, *C* and *D*) opacified material appeared in a large midline vascular channel, which extended from the pulmonary hilar regions through the diaphragm into the abdomen. At the distal end of this channel in the region interpreted as that of the porta hepatis was a globular opacified structure measuring 1.5×2 cm. On the basis of these roentgenograms the diagnosis of total anomalous pulmonary venous connection with the portal system was made.

On the seventh hospital day a thoracotomy was performed on the right side. The anomalous pulmonary venous trunk was easily identified in the posterior mediastinum and was followed to its penetration through the diaphragm. When

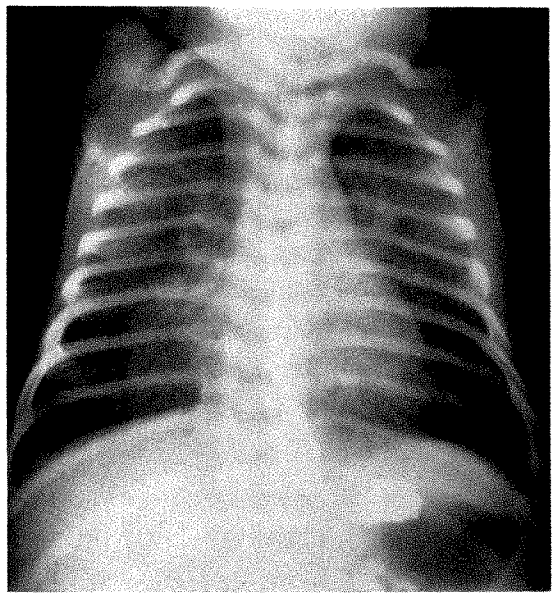


Fig. 4. Case II. Thoracic roentgenogram at 4 weeks shows normal cardiac size and reticular markings in the pulmonary fields.

the pericardium was opened, a small left atrium was identified, with an estimated volume of about 2 cc. Without interruption of venous pathways, the site of convergence of the right pulmonary veins was anastomosed side-to-side to the adjacent wall of the left atrium. The anastomosis was about 1.5 cm. long. At the end of the procedure, the heart stopped, and it was impossible to re-establish a satisfactory beat.

Pathologic Findings. Abnormalities were restricted to the cardiovascular system. The right atrium and right ventricle were dilated and hypertrophied, and the left atrium and left ventricle were somewhat smaller than normal. The aorta and pulmonary artery were normally interrelated. The ductus arteriosus was narrowly patent. The pulmonary veins from the right and left lungs converged in the midline to form a common venous channel. The right aspect of this confluence had been sutured to the left atrium (Fig. 6), forming an opening between the left atrium and the pulmonary veins which admitted a 2 mm. probe. The anomalous common pulmonary venous channel extended downward to the esophageal hiatus anteriorly and to the right of the esophagus and penetrated the diaphragm to enter the abdomen; it terminated by joining a greatly enlarged portal vein (Fig. 7). No pulmonary or systemic veins

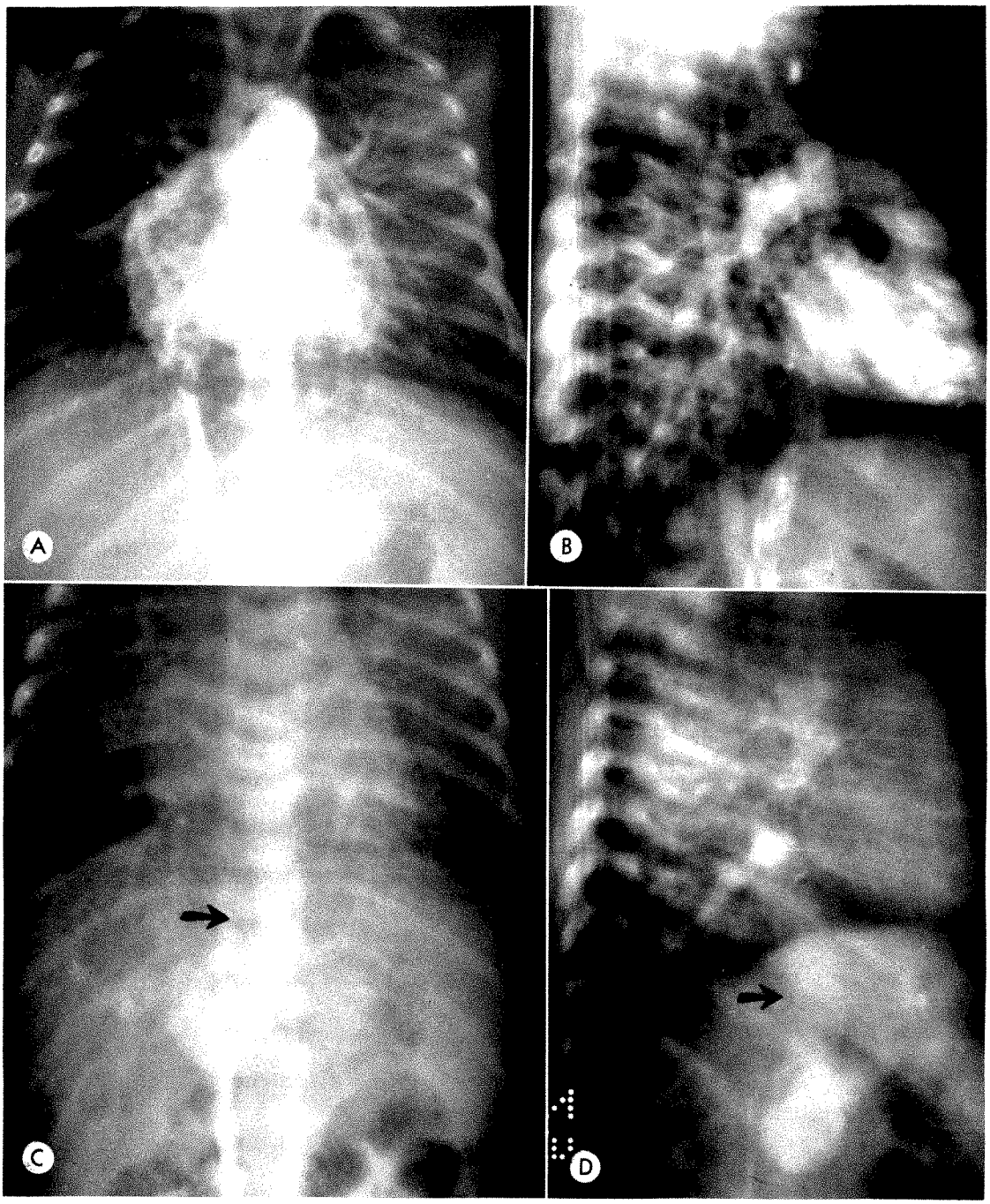


Fig. 5. Case II. Venous angiocardiograms. (*A*) Anteroposterior roentgenogram at 1 second shows the left atrium on each side of the midline, simultaneous opacification of equal density of a large pulmonary artery and a small aorta. (*B*) Lateral roentgenogram at 1 second shows the relative sizes of the pulmonary artery and aorta. (*C*) Anteroposterior roentgenogram at 5 seconds shows opaque material in the common pulmonary venous trunk, which terminates in the region of the porta hepatis. (*D*) Lateral roentgenogram at 5 seconds shows the common pulmonary vein (arrow) and its termination in the region of the porta hepatis.

entered the left atrium (except by way of the surgical anastomosis described). The ventricular septum was intact, and each valve was normal. A valvular competent patent foramen ovale was present. The lungs were observed to be congested.

The inadequate size of the surgical anastomosis between the left atrium and the pulmonary venous system may perhaps be explained by the smallness of the left atrium. It should be pointed out that the operation was performed with the circulation intact. Had artificial circulatory techniques been applied, the opportunity to create a larger opening might have been greater.

CASE III.

Roentgenographic Findings. In a four week old white male infant a thoracic roentgenographic examination was performed in September 1960, during evaluation of cyanosis and dyspnea.

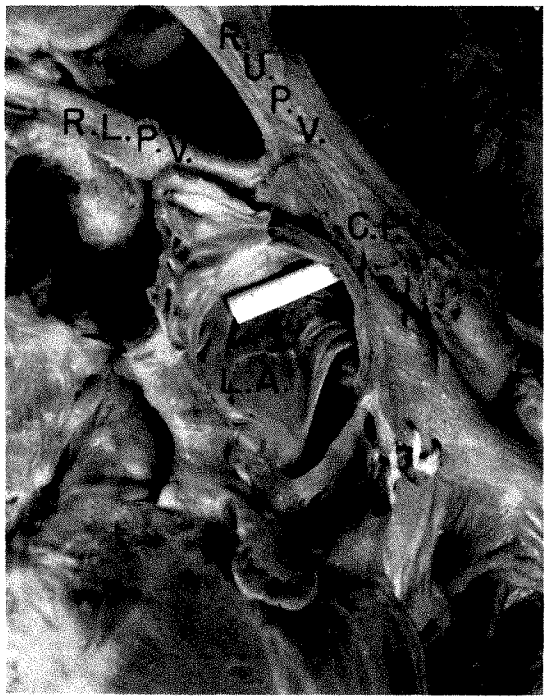


FIG. 6. Case II. Photograph of the gross specimen shows the surgically created anastomosis between the common pulmonary vein (C.P.V.) and the left atrium (L.A.). The probe is lying in the left atrium and is directed through the anastomosis into the common pulmonary vein. The diameter of the surgically created orifice was 2 mm. The right upper pulmonary vein (R.U.P.V.) and right lower pulmonary vein (R.L.P.V.) are seen at the upper left.

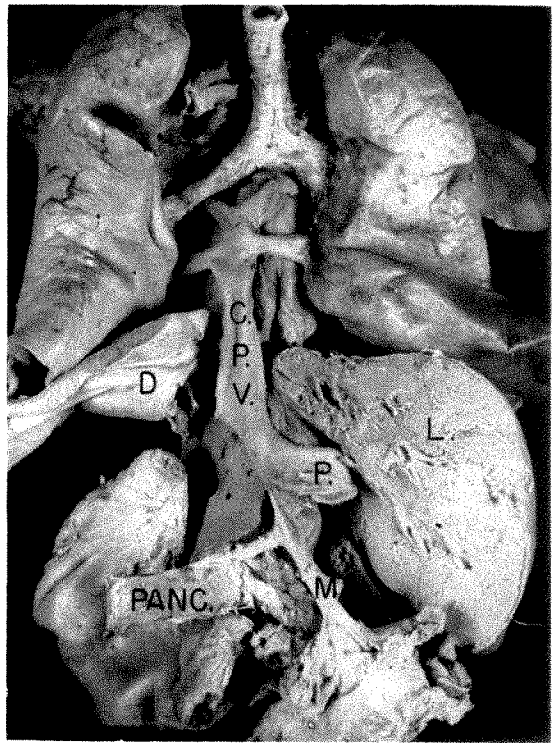


FIG. 7. Case II. Photograph of the specimen of the thoracic and abdominal organs viewed from behind, the esophagus having been removed. The common pulmonary vein (C.P.V.) formed by confluence of the individual pulmonary veins is of large caliber and enters a greatly enlarged portal vein (P.). The superior mesenteric vein (M.) and the splenic vein (above pancreas) are seen forming the portal vein. D.—diaphragm; L.—liver.

This study revealed the cardiac shadow to be of normal size and configuration. The lungs showed diffuse fine stippling bilaterally (Fig. 8). A diagnosis of pulmonary venous obstruction was made.

Venous angiocardiography was performed the following day. Six and one-half cubic centimeters of 90 per cent hypaque-M were injected into the right median basilic vein while films were exposed at two per second for fifteen seconds, in frontal and lateral projections. Following opacification of the right atrium, the left atrium filled, and simultaneous dense opacification of a large pulmonary artery and a small aorta then occurred (Fig. 9, *A* and *B*). Venous return of opacified blood from the lungs was delayed. At five seconds the pulmonary veins filled, and a large channel leading below the diaphragm to the portal vein opacified. (Fig. 10, *A* and *B*).

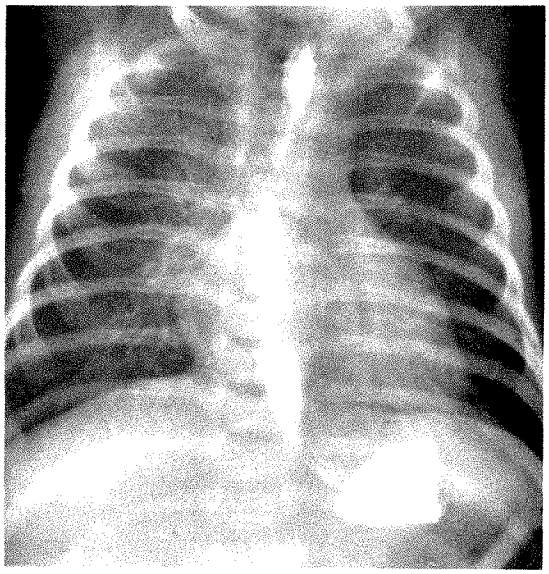


Fig. 8. Case III. Thoracic roentgenogram at 4 weeks shows normal cardiac size and reticular pulmonary markings.

On the sixth hospital day a thoracotomy was performed on the right side. Exploration revealed an anomalous pulmonary venous trunk receiving the venous tributaries from both lungs. This trunk was followed to its penetration of the diaphragm through the esophageal hiatus. When extracorporeal circulation was instituted, severe congestion and edema of the lungs developed. The extracorporeal circulatory procedure was discontinued, and with the circulation intact a side-to-side anastomosis of the anomalous pulmonary venous trunk with the left atrial wall was accomplished. No part of the anomalous pulmonary venous trunk was ligated or divided. Although the patient appeared to tolerate this procedure reasonably well, his condition gradually deteriorated and he died five hours after the operation.

Pathologic Findings. Abnormalities were restricted to the cardiovascular system. The great vessels were normally interrelated. A patent ductus arteriosus having a diameter of 1.4 cm. was noted. The four pulmonary veins joined in the midline to form a common channel that descended anteriorly and a little to the right of the esophagus and penetrated the diaphragm through the esophageal hiatus. This vessel then joined the portal vein (Fig. 10*C*). The surgically performed anastomosis between the left atrium and the common pulmonary venous trunk was identified. The suture line was intact, and the opening measured 4 mm. in diameter (Fig. 11). The ventricular septum was intact. A patent foramen ovale was adjudged to have a potential diameter of 6 mm. The common pulmonary venous trunk was patent throughout, measuring 8 mm. at its narrowest diameter.

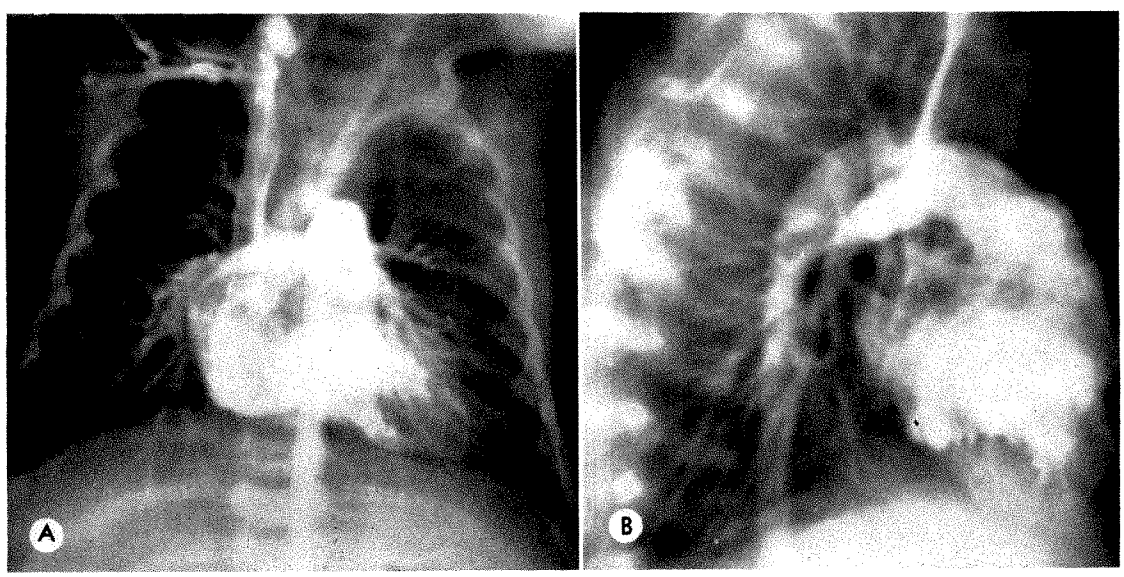


Fig. 9. Case III. Venous angiocardiograms at 1.5 seconds. (*A*) Anteroposterior roentgenogram shows a right-to-left shunt at the atrial level and simultaneous opacification with equal density in the large pulmonary artery and the small aorta. (*B*) Lateral roentgenogram shows opaque material in the left atrium and the enlargement of the pulmonary artery. The descending aorta is clearly evident.

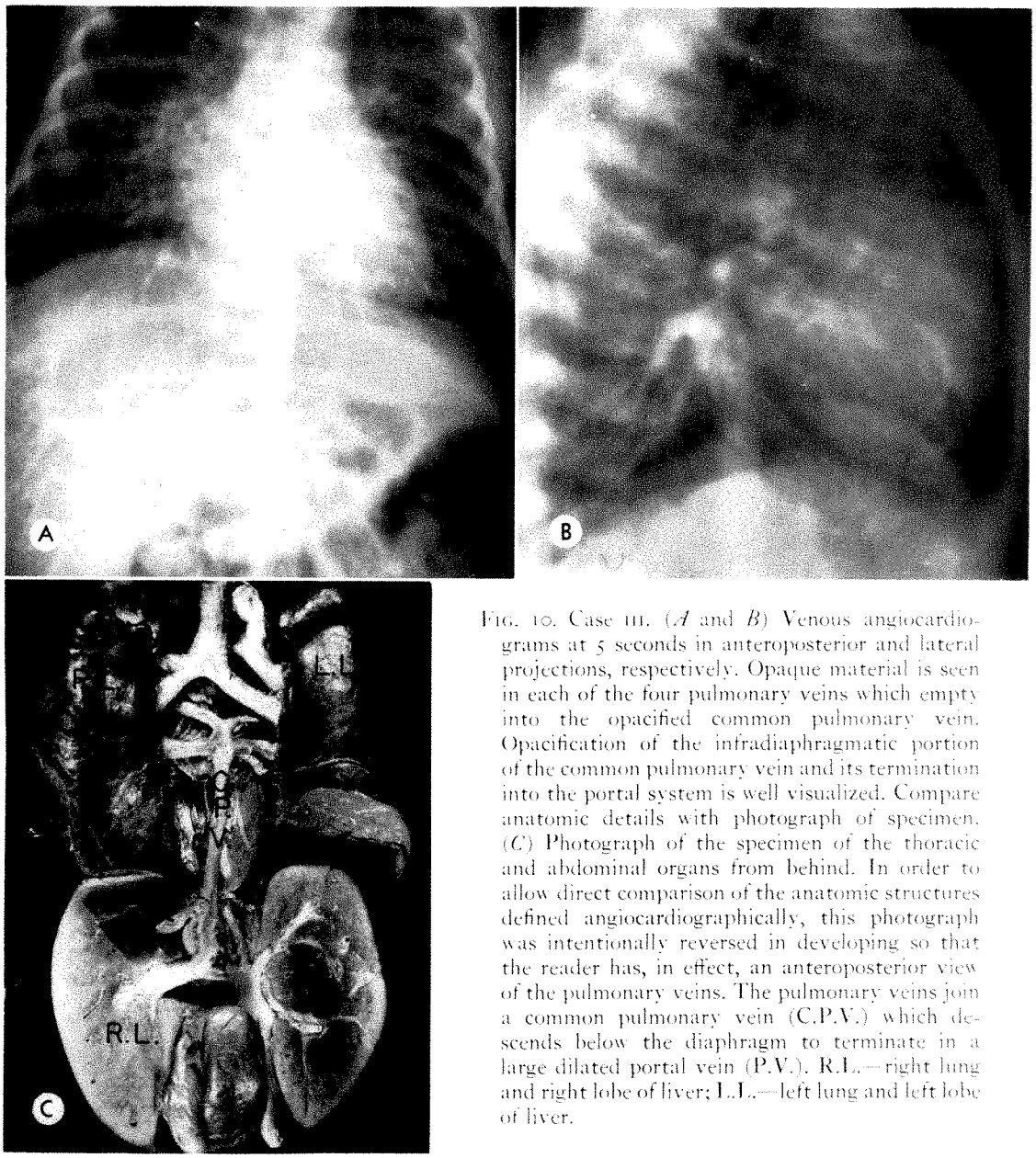


FIG. 10. Case III. (*A* and *B*) Venous angiocardiograms at 5 seconds in anteroposterior and lateral projections, respectively. Opaque material is seen in each of the four pulmonary veins which empty into the opacified common pulmonary vein. Opacification of the infradiaphragmatic portion of the common pulmonary vein and its termination into the portal system is well visualized. Compare anatomic details with photograph of specimen. (*C*) Photograph of the specimen of the thoracic and abdominal organs from behind. In order to allow direct comparison of the anatomic structures defined angiocardiographically, this photograph was intentionally reversed in developing so that the reader has, in effect, an anteroposterior view of the pulmonary veins. The pulmonary veins join a common pulmonary vein (C.P.V.) which descends below the diaphragm to terminate in a large dilated portal vein (P.V.). R.L.—right lung and right lobe of liver; L.L.—left lung and left lobe of liver.

The presence of a large patent ductus arteriosus undoubtedly accounted for the severe congestion of the lungs that developed when extracorporeal circulation was established. This pulmonary insult obviously did not enhance the child's chances for survival.

CASE IV.

Roentgenographic Findings. A thirteen day old white male infant had a thoracic roentgenogram taken in November 1960, during evaluation of cyanosis and dyspnea. Cardiac size and contour were normal and diffuse reticular markings were observed in both pulmonary fields (Fig. 12*A*). Pulmonary venous obstruction was diagnosed, and venous angiocardiography was performed immediately. Six cubic centimeters of 90 per cent hypaque-M were injected into the right saphenous vein while films were exposed in frontal and lateral projections at two per second for fifteen seconds. Opacification of the right atrium was followed immediately by fill-

ing of the left atrium (Fig. 12*B*). Simultaneous dense opacification of a large pulmonary artery and a small aorta then occurred (Fig. 12*C*). Pulmonary veins were visualized at four and one-half seconds (Fig. 12*D*). No reopacification of the left atrium occurred, but, at ten and one-half seconds in the frontal projection, a globular collection of opaque material was seen to the left of the midline between the fifth and sixth ribs (Fig. 12*E*). On the lateral projection (Fig. 12*F*), this shadow lay immediately behind the heart. A faintly opacified channel between the globular shadow and the diaphragm was also seen on the lateral roentgenogram. A diagnosis of anomalous pulmonary venous connection with obstruction was made. The patient died several hours after this procedure and before surgical treatment could be instituted.

Pathologic Findings. Abnormalities were confined to the cardiovascular system. The heart appeared normal to external examination. The pulmonary artery and aorta were normally interrelated, and the ductus arteriosus was closed. No vessels entered the left atrium. The right pulmonary veins entered separately into a muscular walled chamber measuring 1 × 1.5 cm. and having the internal and external appearance of an accessory atrial chamber. Histologically, cardiac muscle was present in the wall of this chamber, which was connected by an atretic strand to the left atrium (Fig. 13, *A* and *B*). The accessory chamber and the two left pulmonary veins converged to form a common venous channel that descended in the midline, pierced the diaphragm through the esophageal hiatus, and terminated in the ductus venosus.

While the common pulmonary venous trunk itself was not narrowed, the upper part of the ductus venosus measured only 1 mm. in diameter. Below the level of entrance of the anomalous pulmonary vein, the ductus venosus was closed; therefore, the only open avenue for escape of pulmonary venous blood was by way of the narrowly patent segment of the ductus venosus into the left hepatic vein. We believe that the accessory atrium-like venous chamber opacified during angiocardiography and presented as a retrocardiac shadow as demonstrated in Figure 12, *E* and *F*.

HISTOLOGY OF THE LUNGS

The lungs were similar histologically in all patients (Fig. 14, *A–D*). Pronounced

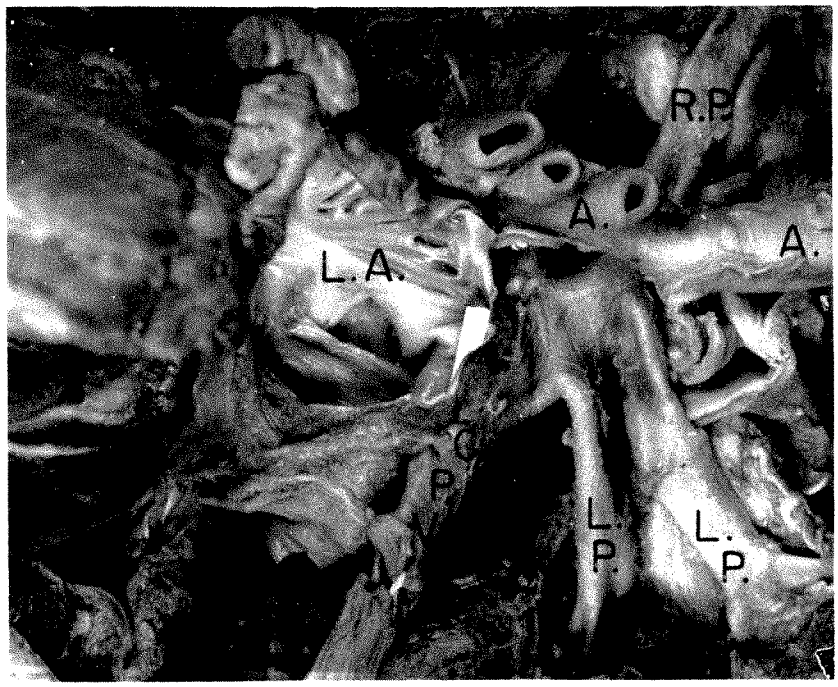


Fig. 11. Case III. Photograph of specimen shows the surgically created anastomosis between the common pulmonary vein (C.P.V.) and the left atrium (L.A.). The probe lies in the left atrium and enters the common pulmonary vein through the anastomosis which measures 4 mm. in diameter. R.P.—right upper and lower pulmonary veins; L.P.—left upper and lower pulmonary veins; A.—aortic arch.

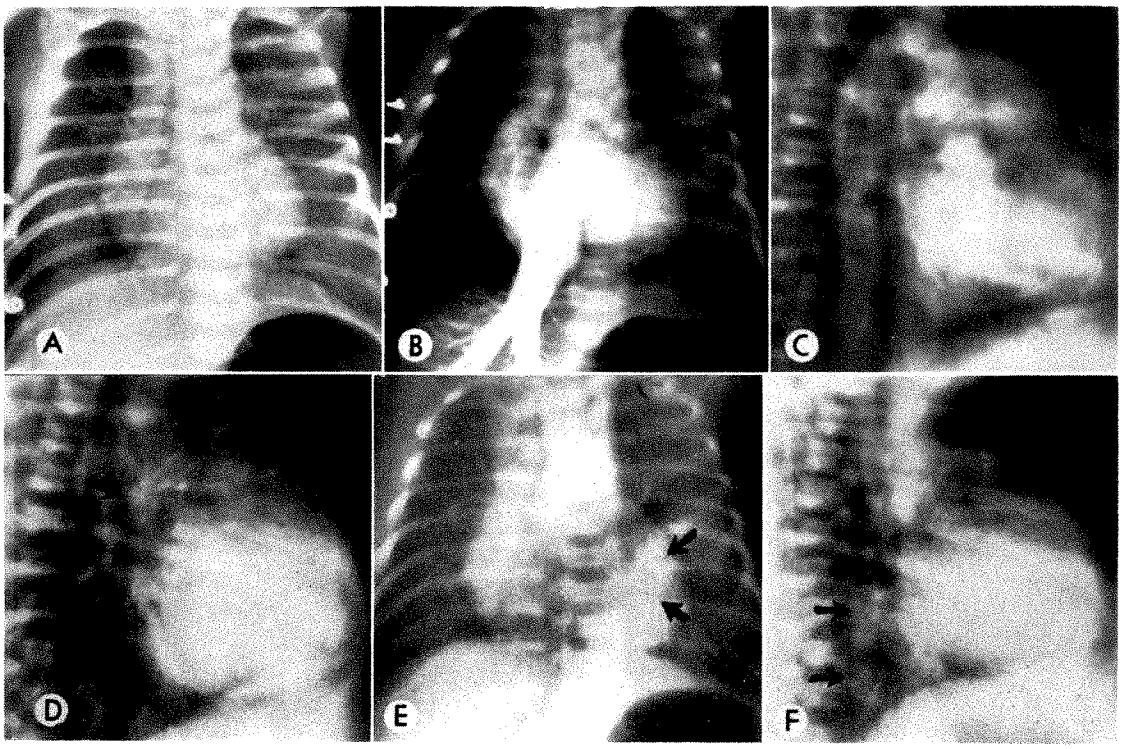


FIG. 12. Case IV. (*A*) Thoracic roentgenogram taken when patient was 13 days of age shows normal cardiac size and reticular pulmonary markings. (*B*) Venous angiocardiogram at 1 second shows right-to-left shunt at atrial level. (*C*) Venous angiocardiogram at 3 seconds shows simultaneous and equal opacification of a large pulmonary trunk and a small aorta. (*D*) Lateral venous angiocardiogram at 4½ seconds shows opacification of pulmonary veins (arrows). (*E*) Venous anteroposterior angiocardiogram at 10½ seconds shows a globular collection of opaque material in relation to the cardiac shadow (arrows). (*F*) Lateral venous angiocardiogram at 10½ seconds shows the globular shadow seen in *E* as lying immediately behind the left atrium. The arrows demonstrate an opacified channel descending toward the diaphragm.

thickening of the alveolar walls resulted from massive capillary dilatation. Hemosiderosis was present, and lymphatic channels in the visceral pleura and interlobular septa were dilated. The pulmonary arterioles revealed pronounced medial hypertrophy in excess of that normally found in this age group. The walls of the pulmonary veins were thickened as a result of very pronounced medial hypertrophy.

DISCUSSION

Total pulmonary venous connection with a vein below the diaphragm is but one of the anomalies possible when no direct communication exists between the left atrium and the pulmonary veins. The more common anomalous connections are between pulmonary veins on one hand, and the left innominate vein, the superior vena cava, coronary sinus, or right atrium on the other. Under these circumstances obstruction to pulmonary venous flow is rare. The clinical and roentgenographic signs in these patients are usually secondary to increased pulmonary blood flow and volume overload of the right ventricle.

When the pulmonary venous blood returns to the right atrium anomalously by way of the portal system of veins, among the factors which combine to increase the resistance to this flow are: (1) the narrow caliber of the common venous trunk and (2) the length of the anomalous route. Interposition of the hepatic capillary bed as in Cases I, II and III or the narrowness of the ductus venosus as in Case IV are additionally contributing factors.

The diagnosis of pulmonary venous obstruction may be suspected from clinical

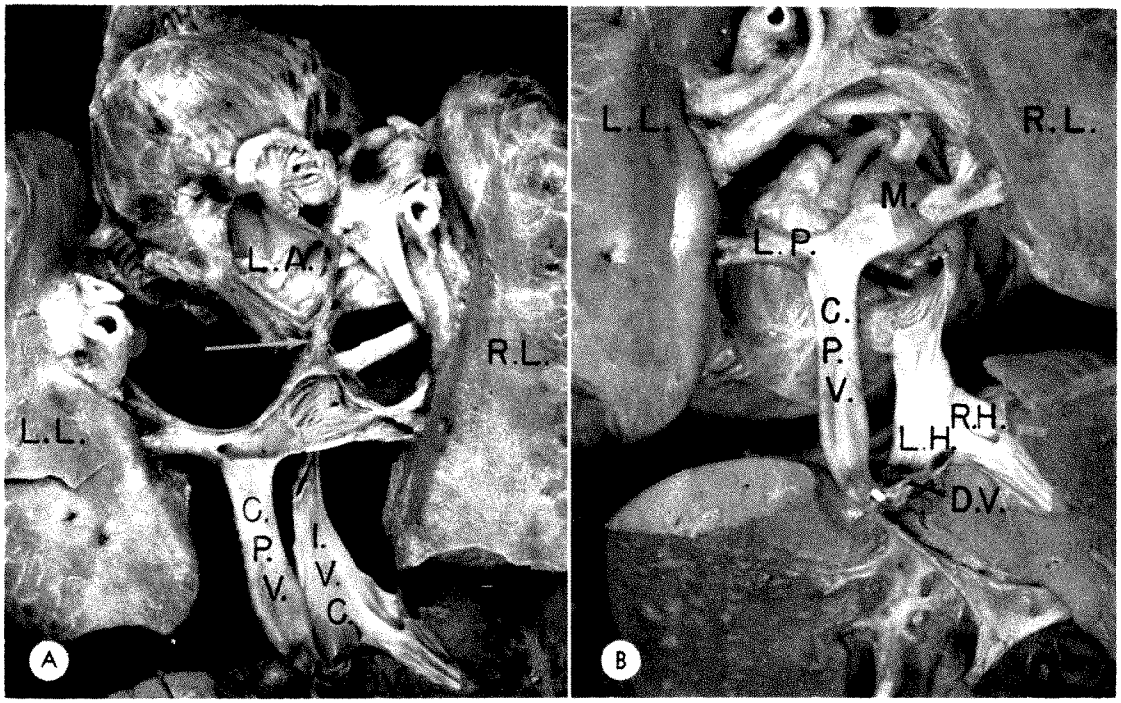


FIG. 13. Case IV. (*A*) Photograph of specimen of the thoracic and abdominal organs viewed from behind. The pulmonary artery and bronchus of the left lung (L.L.) have been severed. The apex of the heart has been elevated. The pulmonary veins from the right lung (R.L.) enter separately into a dilated thick-walled chamber that has the gross texture of an atrial chamber. The only outlet for this chamber is through a common pulmonary vein (C.P.V.) which also receives the veins of the left lung. The anomalous atrial-like chamber was connected to the left atrium (L.A.) by an atretic strand (arrow). The common pulmonary vein terminates below the diaphragm in the ductus venosus (D.V.). The ductus venosus is narrow and terminates in the left hepatic vein. The two hepatic veins join the inferior vena cava (I.V.C.) through which the pulmonary venous blood is finally delivered to the right atrium. (*B*) Photograph of the thoracic organs and liver viewed from behind. The essentials are like those illustrated in (*A*). This view emphasizes the presence of an atrial-like chamber (M.) formed by confluence of the veins from the right lung (R.L.). Histologically, the wall of this chamber was formed mostly of cardiac muscle. L.L.—left lung; L.P.—left pulmonary vein; D.V.—ductus venosus; L.H.—left hepatic vein; R.H.—right hepatic vein.

findings. Thus, any infant in the first few days or weeks of life presenting with cyanosis and dyspnea should be given immediate roentgenographic examination.

Among the findings on the thoracic roentgenogram, a cardiac shadow of normal size with peculiar pulmonary vascular markings should strongly suggest pulmonary venous obstruction. At this point, the differential diagnosis includes the types of pulmonary parenchymal disease occurring not infrequently in the neonatal period. Likewise, pulmonary venous obstruction results from a number of anatomic conditions. These include: (1) cor triatriatum,[15] (2) stenosis or atresia of the pulmonary veins,[12] (3) mitral atresia,[5] and (4) anomalous pulmonary venous connection to other systemic veins through stenotic channels, as well as (5) total anomalous pulmonary venous connection to the portal venous system.

The diagnosis of total anomalous pulmonary venous connection to *any* site, whether or not obstruction to pulmonary venous flow exists, may be made on the basis of the following four signs,[7] each of which was present in every one of our cases: (1) immediate shunt from right atrium to a normal sized or small left atrium; (2) no dilution of the contrast material in the left atrium; (3) simultaneous

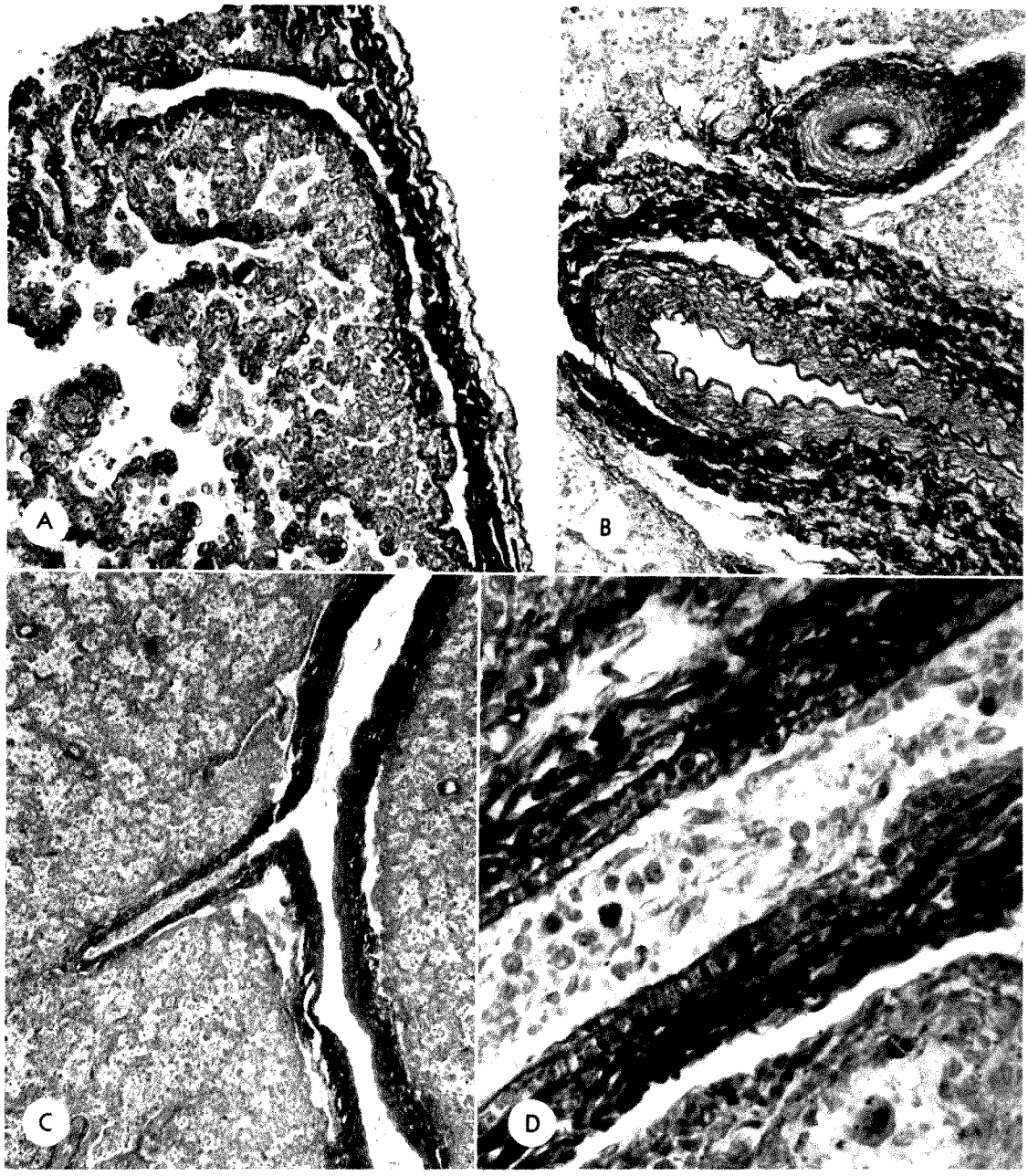


FIG. 14. Photomicrographs of pulmonary vessels from sections stained for elastic tissue. (*A*) Case I. Peripheral portion of lung and visceral pleura show dilatation of a pleural lymphatic and of the pulmonary capillaries (×170). (*B*) Case I. Medial hypertrophy of a large muscular artery and of an arteriole (upper portion of illustration) and an elastic artery are present (×135). (*C*) Case III. A venous trunk receives a tributary. Each shows a thick wall (×150). (*D*) Higher magnification of tributary shown in *C*. The prominence of muscle in the medial layer resembles that seen in arteries. Adventitial prominence is also present (×380).

opacification of an enlarged pulmonary artery and a small aorta; and (4) density of opacification of the aorta equal to or greater than that of the pulmonary artery.

The diagnosis of obstruction in the anomalous venous pathway depends on the foregoing characteristics and, in addition, the signs of: (a) delay in emptying of the

right side of the heart, (b) persistence of opacified blood in the pulmonary vascular system, and (c) failure of reopacification of the left atrium.

Precise anatomic demonstration of the site of anomalous connection of the pulmonary veins with the portal venous system may be established through angiocardiography. The characteristic finding is opacification of the anomalous venous channel with accumulation of contrast medium in the region of the porta hepatis. Low cardiac output and obstruction to pulmonary venous flow may combine to cause late or scant opacification of the anomalous pulmonary venous trunk. Therefore, the angiocardiographic study should be programmed to include roentgenograms taken ten to twelve seconds after the injection is made.

In Case IV, the diagnosis of total anomalous venous connection with obstruction was made on the basis of the presence of each of the aforementioned features. The significance of the opacification shown in Figure 12, *E* and *F* was not fully appreciated until pathologic confirmation.

A helpful clinical sign in each of our 4 cases was intensification of dyspnea and cyanosis during feeding. If one recalls that the anomalous common pulmonary venous trunk accompanies the esophagus through the same hiatus of the diaphragm, this clinical feature may be explained.

In total anomalous connection of the pulmonary veins to the portal venous system, the universal mortality of patients in the first few months of life dictates a prompt attempt to establish a definitive diagnosis. Following diagnosis, immediate surgical correction of the anomaly should be planned. On the basis of our experience to date, we believe that a cardiopulmonary bypass procedure should be utilized to allow the surgeon ample opportunity to make an adequate and accurately placed anastomosis. It is imperative that a patent ductus arteriosus, if present, be ligated prior to the institution of bypass. We feel that the anomalous pulmonary venous trunk should be left patent, since the high resistance imposed by this anomalous route of flow favors drainage through the anastomotic site but at the same time provides a safety valve should the left atrium be unable to accept the whole pulmonary venous return immediately. This pulmonary venous trunk could be ligated later if necessary. Only experience will indicate whether such a channel left open will close spontaneously if the anastomotic connections are adequate.

SUMMARY

1. The universal early mortality in patients with total anomalous venous connection to the portal venous system without major associated cardiac lesions necessitates prompt diagnostic and therapeutic measures.

2. The anatomic features present in this form of anomalous pulmonary venous connection are responsible for pulmonary venous obstruction.

3. On the basis of the clinical, physical, electrocardiographic, and routine roentgenographic findings, a diagnosis of pulmonary venous obstruction may be made in these patients.

4. Angiocardiography provides confirmation of the pulmonary venous obstruction and allows a definitive anatomic diagnosis of total anomalous pulmonary venous connection to the portal venous system.

5. Attempts to correct this condition surgically in 2 of 4 patients met with failure, but on the basis of this experience, it is believed that surgical correction of this defect remains feasible.

Richard G. Lester, M.D.
Department of Radiology
Medical College of Virginia
Richmond 19, Virginia

REFERENCES

1. BOR, I. Anomální vústěni plichních zil do jaternich zil. *Pediat. Listy*, 1953, *8*, 152.
2. BURROUGHS, J. T., and EDWARDS, J. E. Total anomalous pulmonary venous connection. *Am. Heart J.*, 1960, *59*, 913–931.

3. Butler, H. Abnormal disposition of pulmonary veins. *Thorax*, 1952, *7*, 249–254.
4. Darling, R. C., Rothney, W. B., and Craig, J. M. Total pulmonary venous drainage into right side of heart. *Lab. Invest.*, 1957, *6*, 44–64.
5. Edwards, J. E., and DuShane, J. W. Thoracic venous anomalies. *Arch. Path.*, 1950, *49*, 517–537.
6. Ghon, A. Ein Beitrag zu den Anomalien der Pulmonalvenen. *Beitr. path. Anat.*, 1916, *62*, 175–193.
7. Gott, V. L., Lester, R. G., Lillehei, C. W., and Varco, R. L. Total anomalous pulmonary venous return; analysis of 30 cases. *Circulation*, 1956, *13*, 543–552.
8. Harris, G. B. C., Neuhauser, E. B. D., and Giedion, A. Total anomalous pulmonary venous return below diaphragm; roentgen appearances in three patients diagnosed during life. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 436–441.
9. Johnson, A. L., Wiglesworth, F. W., Dunbar, J. S., Siddoo, S., and Grajo, M. Infradiaphragmatic total anomalous pulmonary venous connection. *Circulation*, 1958, *17*, 340–347.
10. Keith, J. D., Rowe, R. D., Vlad, P., and O'Hanley, J. H. Complete anomalous pulmonary venous drainage. *Am. J. Med.*, 1954, *16*, 23–38.
11. Laurence, K. M., and Brown, R. J. K. Total anomalous drainage of pulmonary veins into left gastric vein. *Brit. Heart J.*, 1960, *22*, 295–299.
12. Lucas, R. V., Jr., Anderson, R. C., Woolfrey, B. F., and Edwards, J. E. Atresia of common pulmonary vein. To be published.
13. Munck, W. Über abnormen Verlauf der Lungenvenen. *Acta path. et microbiol. scandinav.*, 1933, *10*, 321–329.
14. Mykschowszky, G. Zur Kenntnis der Anomalien der Lungenvenen. *Klin. med. Wien.*, 1948, *3*, 263–269.
15. Niwayama, G. Cor triatriatum. *Am. Heart J.*, 1960, *59*, 291–317.
16. Parsons, H. G., Purdy, A., and Jessup, B. Anomalies of pulmonary veins and their surgical significance: report of three cases of total anomalous pulmonary venous return. *Pediat.*, 1952, *9*, 152–166.
17. Rosenfeld, I., Silverblatt, M. L., and Strauss, L. Total anomalous pulmonary venous drainage into portal vein. *Am. Heart J.*, 1957, *53*, 616–623.
18. Sherman, F. E., and Bauersfeld, S. R. Total, uncomplicated, anomalous pulmonary venous condition: morphologic observations on 13 necropsy specimens from infants. *Pediat.*, 1960, *25*, 656–668.
19. Smith, B., Frye, T. R., and Newton, W. A. Total anomalous pulmonary venous return; infradiaphragmatic type, clinical manifestations. Society for Pediatrics Research. Abstracts, 29th Annual Meeting, 1959, p. 69.

CONGENITAL PULMONARY LYMPHANGIECTASIS*

REPORT OF A CASE WITH ROENTGEN FINDINGS

By R. W. CARTER, M.D., *and* H. M. VAUGHN, M.D.
BIRMINGHAM, ALABAMA

CONGENITAL pulmonary lymphangiectasis, although relatively rare, has been fairly frequently the subject of papers appearing in the pathologic literature. It has been variously termed "congenital lymphangiomatosis of the lung,"[2] "congenital pulmonary cystic lymphangiectasis,"[1,3] and "congenital dilatation of pulmonary lymphatics."[5] Twenty-one cases have been reported in the world literature according to Frank and Piper[1] in their article published in 1959; however, we have been unable to find any description of this condition in the radiologic literature.

The cases previously described, as well as the present case, appear to have a fairly uniform clinical picture: that of neonatal respiratory distress and cyanosis with death occurring in a few hours or a few days. Some cases have also been observed in stillborns. Most often, the delivery and gestation have been uneventful.

Of the cases reported, we have been able to find only 2 of which roentgenograms were made. These, in addition to the present case, apparently represent the only cases of congenital pulmonary lymphangiectasis which have been brought to the attention of the radiologist. Because of the clinical similarity of this disease to other conditions more familiar to the radiologist, we feel that our patient in whom the roentgenographic findings could not be distinguished from those of hyaline membrane disease or fetal atelectasis is worthy of reporting. Maidman and Barnett[5] have stated, "It is apparent from a review of all cases that this entity . . . must be included with other disease processes responsible for neonatal cyanosis and, unfortunately, can be positively identified only by means of autopsy."

The roentgenograms of the 2 previously reported cases appear to be similar to those of the present case, although the changes are probably more marked. The findings were described as "bilateral multiple scattered densities,"[5] and "an enlarged heart and increased opacity of both lung fields with some mottling suggestive of diffuse cystic change." In the latter case, the heart appeared "somewhat dilated" at necropsy, so definite cardiomegaly may not have been present.[4]

REPORT OF A CASE

On November 1, 1960, at 10:45 P.M., the patient was born at full term by low forceps delivery. He weighed 7 pounds and 6 ounces. At birth, the heart rate was rapid, respirations were rapid and shallow, and the infant was cyanotic. The liver was thought to be enlarged. The infant was placed in a rocking-bed respirator and given moist oxygen.

A portable anteroposterior chest roentgenogram made approximately twelve hours following birth showed a normal heart, widening of the anterior and superior mediastinal regions, indicating a thymus gland, fine granular densities distributed throughout both lungs and a normal position of the diaphragm. The bony thorax showed no abnormality. The diffuse granular pulmonary densities, coupled with the clinical history, were thought to represent hyaline membrane disease (Fig. 1, *A* and *B*).

About fourteen hours after birth, the infant was digitalized and a liquefying agent (Chymar aqueous) was given. Penicillin therapy was also started. The infant failed to respond and expired approximately sixteen hours after birth.

At autopsy, the liver was found to be grossly enlarged, extending into the left side of the abdomen. The sinusoids were dilated and filled

* From the Department of Radiology, The Norwood Clinic and Carraway Methodist Hospital, Birmingham, Alabama.

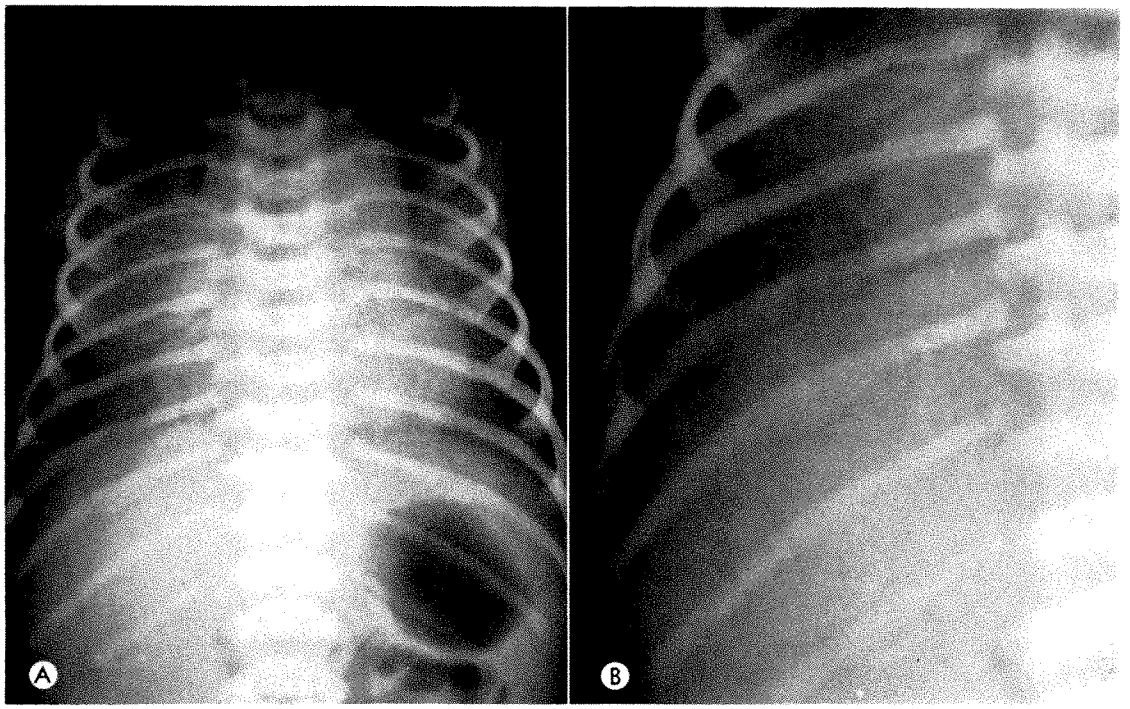


FIG. 1. (*A*) Fine granular densities are distributed throughout both lungs, representing markedly dilated cystic lymphatics. This picture cannot be distinguished from that in hyaline membrane disease or in fetal atelectasis. (*B*) Detail of the right lower lung field shows the diffuse granular densities.

with blood. Scattered throughout the liver were numerous small collections of immature blood cells. The thymus was of normal size and showed numerous areas of hemorrhage which were relatively small, although in some instances, the hemorrhage became confluent and covered a fairly large area. The heart was normal.

Both lungs were of normal size. The pleural surfaces were smooth and glistening. The surface and cut surface of both lungs were of a healthy pink color. Microscopic examination showed the alveolar spaces to be moderately enlarged. In the interstitial tissue and particularly just beneath the pleura, the lymphatics were markedly dilated. This picture was noted in all of the sections of lung studied microscopically. In some areas, the alveolar spaces contained a few squames; however, there was very little inflammatory reaction at any point in the lungs. No evidence of hyaline membrane disease was found (Fig. 2).

A physiologic patent ductus arteriosus and patent foramen ovale were noted. The cranial cavity was not entered.

The final anatomic diagnosis was (1) congenital pulmonary lymphangiectasis, (2) passive congestion of the liver, (3) petechiae of the thymus, (4) extramedullary hematopoiesis of the liver, (5) patent foramen ovale (physiologic), and (6) patent ductus arteriosus (physiologic.)

COMMENT

Congenital pulmonary lymphangiectasis is a condition occurring in newborns and to

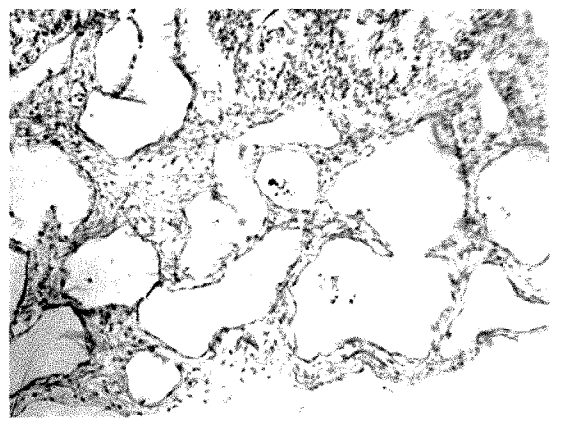


FIG. 2. Photomicrograph showing the dilated lymphatics in the lung.

a lesser extent in stillborns. In most instances gestation and delivery are normal but the child commences to have respiratory distress and cyanosis within minutes or a few hours after birth. Because of the increasing frequency with which roentgenography of the newborn in respiratory distress is performed, it is important that the radiologist be aware of this disease and its roentgen similarity to more familiar conditions. Although we have been unable to find a description of the roentgen findings in the radiologic literature, it is likely that this condition is much more common than previously realized, and that many cases formerly diagnosed as congenital heart disease, hyaline membrane disease, fetal atelectasis, etc., and which were not pathologically confirmed may have represented this condition. It is anticipated that this diagnosis will be made more frequently in the future.

SUMMARY

1. The first case of congenital pulmonary lymphangiectasis reported in the radiologic literature is presented.

2. The clinical symptoms of congenital pulmonary lymphangiectasis simulate hyaline membrane disease and some types of congenital heart disease.

3. The roentgenographic findings in congenital lymphangiectasis of fine granular densities distributed throughout the lungs simulate those of hyaline membrane disease and fetal atelectasis.

R. W. Carter, M.D.
The Norwood Clinic
1529 North 25th Street
Birmingham 4, Alabama

REFERENCES

1. FRANK, J., and PIPER, P. G. Congenital pulmonary cystic lymphangiectasis. *J.A.M.A.*, 1959, *171*, 1094–1098.
2. GIAMMALVO, J. T. Congenital lymphangiomatosis of lung: form of cystic disease; report of case with autopsy findings. *Lab. Invest.*, 1955, *4*, 450–456.
3. LAURENCE, K. M. Congenital pulmonary cystic lymphangiectasis. *J. Path. & Bact.*, 1955, *70*, 325–333.
4. LAURENCE, K. M. Congenital pulmonary lymphangiectasis. *J. Clin. Path.*, 1959, *12*, 62–69.
5. MAIDMAN, L., and BARNETT, R. N. Congenital dilatation of pulmonary lymphatics. *Arch. Path.*, 1957, *64*, 104–106.

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ESOPHAGEAL STRICTURE FOLLOWING INGESTION OF CLINITEST TABLETS*

By JACK G. RABINOWITZ, M.D., *and* STANLEY TCHANG, M.D.
BROOKLYN, NEW YORK

ESOPHAGEAL strictures following the accidental ingestion of Clinitest tablets have recently been described in the pediatric and otolaryngologic literature. These commercially available tablets are used at home by patients with diabetes to test for glycosuria. The tablets contain anhydrous sodium hydroxide and therefore represent a new type of lye ingestion hazard. Since the Clinitest stricture has certain characteristic features, a report of such a case in the radiologic literature appears justifiable.

REPORT OF A CASE

A sixteen year old girl was admitted to the Kings County Hospital on July 13, 1959, one day after she inadvertently swallowed two Clinitest tablets, used by her father, a diabetic. She had mistaken them for aspirin. Immediately after swallowing the pills the patient felt a burning sensation substernally and regurgitated the partially dissolved tablets. Inasmuch as the burning sensation persisted the patient was admitted for observation. There were no abnormal physical findings. Esophagoscopic examination on July 15, 1959 revealed only minimal congestion of the esophagus. She was treated for her symptoms, improved clinically and left the hospital two days later. One week thereafter she began to have difficulty swallowing solid foods. She experienced a sensation of heaviness in the chest during and after meals. This was associated with occasional vomiting. She returned to the hospital and a roentgenologic examination of the esophagus on July 30, 1959, revealed a short, well circumscribed constriction at the junction of the middle and lower thirds of the esophagus (Fig. 1*A*). A small ulcer crater arose from the posterior aspect of the constricted area (Fig. 1*B*). The esophagus was not dilated proximal to the lesion and there was no delay in the descent of the thick barium mixture. Exophagoscopic examination revealed a short ring-like stricture approximately 33 cm. from the upper incisor teeth. Stretching with Hurst dilators was performed almost daily until a No. 28 dilator was passed. An esophagram obtained on August 10, 1959 showed some widening of the previously noted stricture and no ulceration was noted. The patient had no further difficulty in swallowing solid foods and was discharged from the hospital on August 13, 1959.

PATHOGENESIS

Caustic urine-sugar reagent tablets (Clinitest) contain sodium hydroxide, sodium bicarbonate, citric acid and copper sulfate. Sodium hydroxide liberates a large amount of heat on hydration when placed in solution and if this occurs in the esophagus a thermal burn of the mucosa results. The caustic action of sodium hydroxide causes direct necrosis. Copper sulfate is both astringent and irritant.[3,10,12]

Diffuse edema is the initial pathologic effect of lye ingestion. Occasionally, this may be demonstrated by an esophagram. However, roentgenologic studies performed during this phase in the previously reported cases of Clinitest tablet ingestion have revealed no changes of diagnostic or prognostic significance.[2,10] Liquefaction necrosis follows rapidly with sloughing of the necrotic material. Repair begins at about the end of the second week and the resulting fibrosis leads to stricture formation.

Since the accident under consideration in all likelihood involves the arrest of a firm tablet during its passage through the esophagus, it is quite understandable that the resultant stricture is short and ring-

* From the Departments of Radiology, State University of New York Downstate Medical Center and the Kings County Hospital Center, Brooklyn, New York.

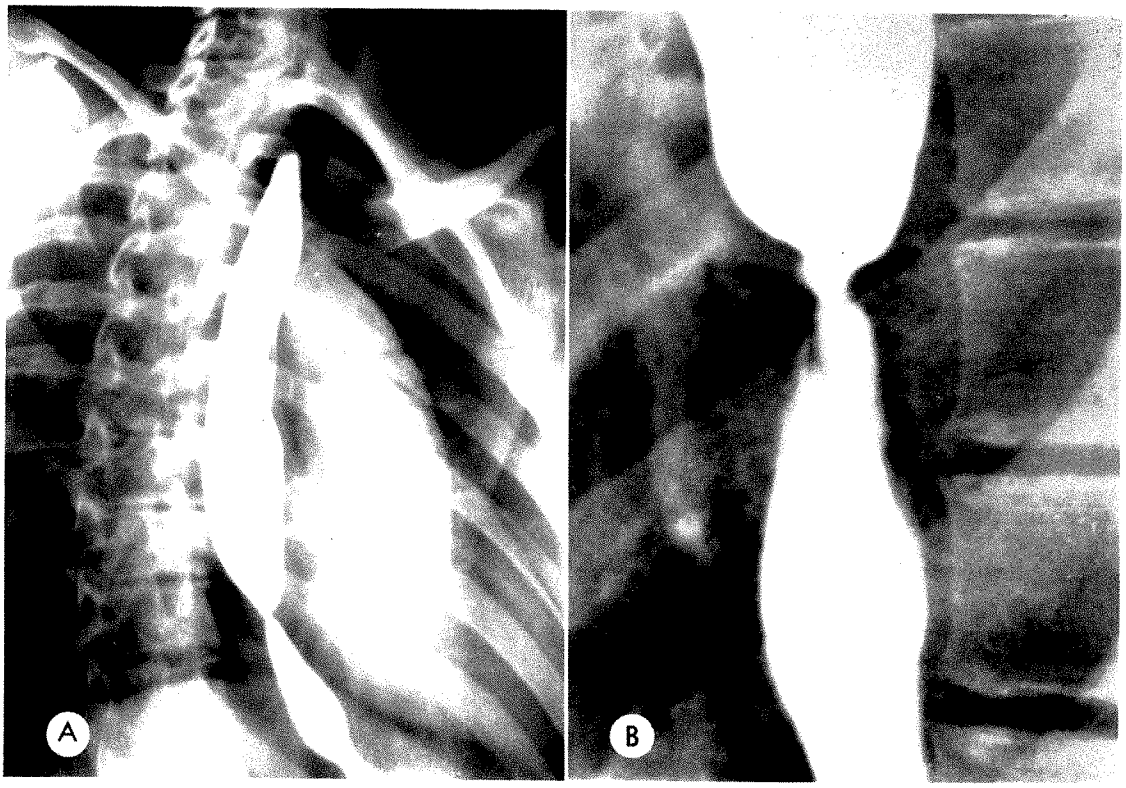


FIG. 1. (*A*) Roentgenogram demonstrates the short, ring-like stenosis of the esophagus at approximately the junction of the middle and lower thirds. The esophagus proximal and distal to this area is normal. (*B*) Spot roentgenogram of the strictured area shows the small ulcer crater arising from the posterior portion of the esophageal wall.

like. This differs from the usual type of lye ingestion—drinking a caustic solution—in which the stricture is usually elongated and has tapered ends. Arrest of the tablet can apparently occur at any of the usual areas of anatomic or physiologic narrowing particularly if only small amounts of water are ingested with the tablet. The previous case reports in the literature describe the occurrence of strictures in all portions of the esophagus. No changes need be produced if there is sufficiently rapid passage of the tablet through the esophagus or if an adequate amount of fluid is ingested simultaneously. One patient has been reported to have taken one tablet four times a day for several weeks. However, each tablet was swallowed with a full glass of water and no esophageal or gastric disorders were noted on follow-up examinations.[10] The stomach usually remains uninjured because of the neutralizing effect of the acid gastric secretions and the protective effect of gastric mucin.

ROENTGEN FINDINGS

The diagnosis of caustic tablet (e.g., Clinitest) stricture should be thought of when there is a smooth-walled, sharply-circumscribed, short ring-like constriction of the esophagus in an otherwise normal esophagus (Fig. 1, *A* and *B*). Because ulcer formation within the stricture is quite common, the lesion must be distinguished from peptic esophagitis. The latter is most frequently seen in the lower third of the esophagus and is often associated with an hiatus hernia. Ulceration arising in heterotopic gastric mucosa may produce the radiating mucosal folds so commonly found in gastric ulcers. The constricting annular "lower esophageal ring" should be easy to

distinguish because of its location at the gastroesophageal mucosal junction and its typical diaphragm-like appearance.[4] Diffuse systemic sclerosis with esophageal stricture formation has the characteristics of a chronic diffuse esophagitis. The presence of characteristic changes in peristalsis and in the emptying mechanism together with scleroderma involving the hands, feet or face should lead to the correct diagnosis. Epidermolysis bullosa produces an esophageal stricture which is usually smooth, somewhat lengthy and tapers at both ends. It is always accompanied by cutaneous manifestations.[1] Congenital esophageal webs are usually located on the anterior wall of the esophagus at the level of the cricoid cartilage. Conceivably, an infiltrative neoplasm could simulate a benign stricture but since most of the reported cases are in children the differential diagnosis of a neoplasm may arise in only the occasional case found in adults.

CLINICAL COURSE

Of the 7 cases thus far reported in the literature, 5 have been in children. The clinical course following the ingestion of Clinitest tablets is quite similar to that following ordinary lye ingestion. Early cortisone and antibiotic therapy has been recommended to prevent stricture formation.[8] Treatment in our case has been limited essentially to bougienage. Whether more dilatation will be required in the future cannot be determined at this time. Where persistent narrowing existed, simple resection with end to end anastomosis has been successful.[2,10,12]

SUMMARY

Caustic urine-sugar reagent tablets (Clinitest tablets) are widely used by the laity and cases of accidental ingestion continue to occur. The radiologist should be aware of the characteristic smooth-walled, sharply circumscribed, short ring-like stricture which may be produced.

Jack G. Rabinowitz, M.D.
Department of Radiology
State University of New York
Downstate Medical Center
Brooklyn 3, New York

REFERENCES

1. ALPERT, M. Roentgen manifestations of epidermolysis bullosa. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1957, *78*, 66–72.
2. BLOOMER, W. E., and KIRCHNER, J. A. Esophageal stricture due to Clinitest. *Connecticut M. J.*, 1955, *19*, 91–92.
3. CANBY, J. P. Clinitest produced esophageal stricture. *J. Pediat.*, 1957, *50*, 68–70.
4. MACMAHON, H. E., SCHATZKI, R., and GARY, J. E. Pathology of lower esophageal ring; report of case, with autopsy, observed for nine years. *New England J. Med.*, 1958, *259*, 1–8.
5. MCFADDEN, R. L. Stricture of esophagus due to accidental ingestion of Clinitest tablet. *Journal-Lancet*, 1958, *78*, 533–534.
6. MELLINS, H. Z. Esophageal ulcer in infancy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1952, *68*, 634–638.
7. OWENS, H. Chemical burns of esophagus: Importance of various chemicals as etiologic agents in stricture formation. *A.M.A. Arch. Otolaryng.*, 1954, *60*, 482–486.
8. RAY, E. S., and MORGAN, D. L. Cortisone therapy of lye burns of esophagus. *J. Pediat.*, 1956, *49*, 394–397.
9. STOTHERS, H. H. Chemical burns and strictures of esophagus. *A.M.A. Arch. Otolaryng.*, 1952, *56*, 262–276.
10. TOMSOVIC, E. J., and JAVID, H. Cicatricial stenosis of esophagus following ingestion of single urine-sugar reagent tablet. *J. Pediat.*, 1958, *53*, 608–614.
11. VARGAS, L. L., BRITTON, R. C., and GOODMAN, E. N. Congenital esophageal stenosis; report of case of annular muscle hypertrophy at esophagogastric junction. *New England J. Med.*, 1956, *255*, 1224–1227.
12. ZIMMERMAN, C. Esophageal stricture from accidental ingestion of Clinitest tablets. *A.M.A. J. Dis. Child.*, 1959, *97*, 101–103.

ANOMALOUS POSITION OF THE FOREGUT*

By BERTA M. RUBINSTEIN, M.D., HOWARD BLOOM, M.D., REGINA GLUCK, M.D., *and* MILTON J. H. GRAND, M.D.
NEW YORK, NEW YORK

MANY excellent reviews on malrotation of the bowel have appeared in recent publications, but these have dealt for the greater part with abnormalities in the embryologic positioning of the midgut portion of the primitive gastrointestinal tract. Little has been said, regarding the anomalous position of the foregut and its anlage. The case presented is a two and one-half year old white girl with congenital heart disease. While undergoing routine roentgenologic examination for this condition, she was found to have an abnormally located stomach, which at first suggested situs inversus, but was later shown to be a case of failure of rotation of the foregut.

REPORT OF A CASE

This two and one-half year old female was first seen at Montefiore Hospital on March 16, 1959 for investigation of congenital heart disease. Her birth weight on March 3, 1956 was 7 pounds 15 ounces. Cyanosis was noted shortly after birth and vomiting occurred at the same time. Roentgen findings at that time indicated an "upside down stomach with partial obstruction." She was transferred to Meadowbrook Hospital at the age of one week, and repeat roentgenograms indicated "situs inversus abdominalis with no obstruction." The vomiting was controlled by careful feeding, and the gastrointestinal disturbances subsided. At the age of two weeks she went into acute cardiac failure which was controlled with digitalis. She was discharged at the age of one month, when she had regained her birth weight. The patient did well at home except for persistence of slight cyanosis. At the age of five months, following an episode of diarrhea, the cyanosis increased and she went into syncope during crying spells. She suffered no further episodes of cardiac embarrassment, but she developed slowly, and by the age of two years, she could not walk without support and weighed only 18 pounds 10 ounces. In June, 1958, at the age of twenty-six months, she developed a left hemiplegia and congestive heart failure. She was digitalized, and put on mercurials and salt restriction, but her condition did not clear up, and she was admitted to Montefiore Hospital for further investigation.

Physical examination revealed an underdeveloped girl who had resting cyanosis, clubbing of the fingers and toes and peripheral edema. Rales were heard in both lungs and fluid was present. The liver was palpated 6 cm. below the right costal margin. The blood pressure was 90/60, and the heart rate was regular at 120 per minute with a loud systolic murmur, heard best in the left fourth interspace at the sternal edge. A precordial bulge was evident and the heart was enlarged to percussion. On neurologic examination no residual of her earlier hemiplegia was found.

Laboratory Data. Hematocrit was 66 per cent, the hemoglobin was 20 gm. per cent, the red blood cell count was 7,230,000 per cu. mm., the white blood cell count was 8,900 per cu. mm. with 79 per cent polymorphonuclears, 20 per cent lymphocytes and 1 per cent monocytes. The platelet count was 170,000 per cu. mm. Specific gravity of the urine was 1.009 with a trace of albumin but without sugar or white cells. An electrocardiogram revealed right ventricular hypertrophy.

Roentgen Findings. Fluoroscopy showed a bizarre cardiac shadow consistent with transposition of the great vessels. Roentgenography with barium in the esophagus (Fig. 1 and 2) disclosed marked enlargement of the cardiac silhouette with the left border reaching almost to the left lateral chest wall. These studies and roentgenograms of the upper gastrointestinal tract showed the stomach to be located in the right side of the abdominal cavity with the greater curvature on the right and the lesser curvature on the left (Fig. 1 and 3). The pylorus

* From the Divisions of Diagnostic Radiology and Laboratories and the Department of Pediatrics, Montefiore Hospital, New York, New York.

and first part of the duodenum pointed across the midline to the left and anteriorly (Fig. 1, 2 and 4). The liver, however, was in the right upper quadrant (Fig. 3). On this basis, the diagnosis of failure of rotation of the foregut was made, rather than situs inversus abdominalis.

A selective angiocardiogram using 70 per cent Urokon outlined a large left ventricle, which gave rise to the aorta, situated to the left of the left pulmonary artery. The right and left pulmonary arteries were well visualized, but the main pulmonary artery could not be clearly delineated.

In view of the inability to establish a definitive diagnosis, a second injection was made into the left ventricle since the catheter could easily be passed through a large interatrial septal defect. During this second procedure, the child developed ventricular extrasystoles and then went into ventricular fibrillation with cardiac arrest. She did not respond to cardiac massage and died.

Necropsy Findings. At necropsy performed five hours after death, 100 cc. of clear yellow fluid was found in the peritoneal cavity. The position of the esophagus was unremarkable and it terminated in the cardia of the stomach at about 1 cm. beyond the diaphragmatic hiatus. The stomach lay within the right upper quadrant of the abdominal cavity with the cardia and greater curvature lying on the right while the lesser curvature was on the left. The pylorus pointed across the midline from the right side and the first part of the duodenum pointed anteriorly. The duodenum then continued to form a normal loop which was, however, concave to the right and terminated at the duodenojejunal junction which lay in the right upper quadrant. The jejunum and ileum were completely patent but were abnormally located, lying almost completely in the right lower quadrant of the abdominal cavity. The terminal ileum ascended in the midline up toward the diaphragm and entered the cecum

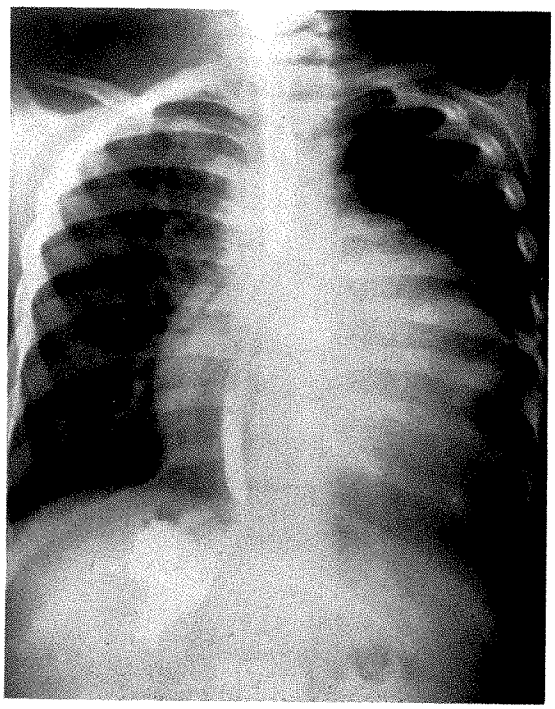


FIG. 1. Posteroanterior chest roentgenogram with the esophagus and upper stomach opacified with barium shows the heart markedly enlarged in its transverse diameter. The lower half of the esophagus is bowed to the right and the stomach is seen well to the right of the midline.

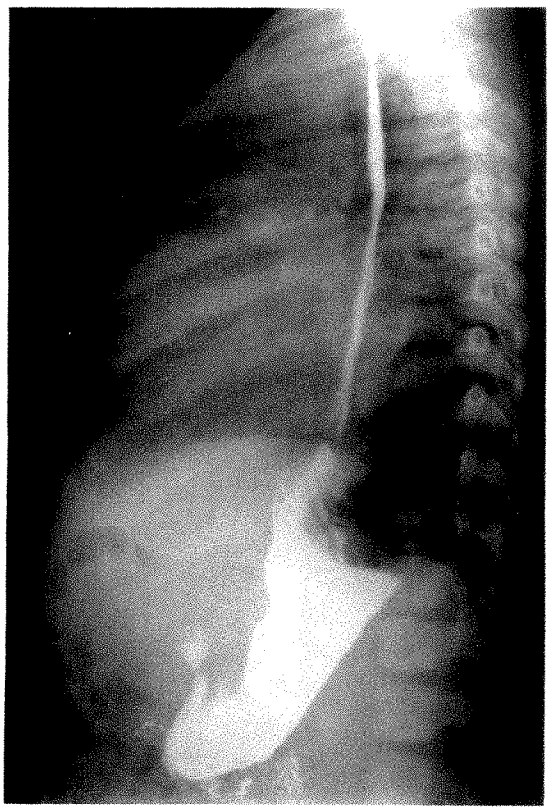


FIG. 2. A lateral roentgenogram of the chest with the esophagus, stomach and duodenal bulb opacified with barium shows the pylorus and duodenal cap pointed forward. Gas is seen in the postbulbar duodenal segment which is far anterior to its usual position.

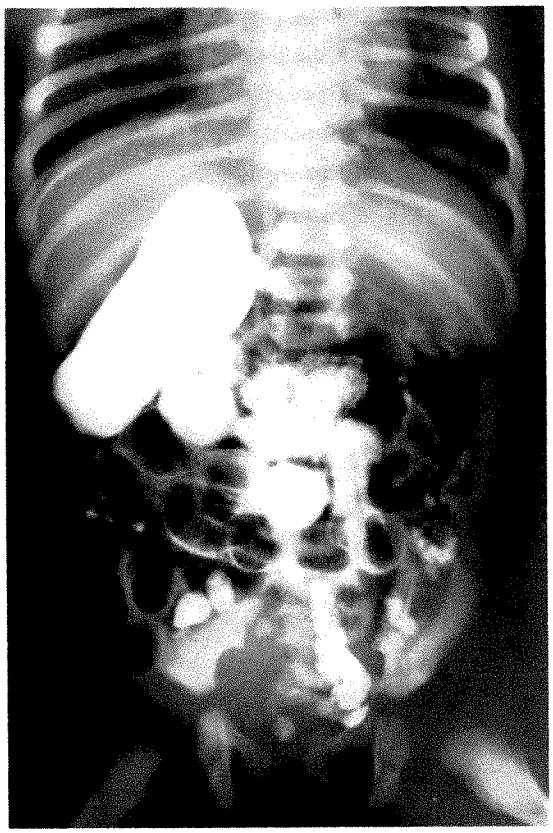


FIG. 3. A frontal plane roentgenogram made during an upper gastrointestinal tract examination shows the barium-filled stomach to be well to the right of the midline. The greater curvature of the stomach is on the right side. The entire small bowel lies mostly on the right side of the abdomen. An opacity representing the liver is seen in the right upper quadrant.

at this point. The cecum, appendix and large bowel were unremarkable in appearance with no evidence of diverticula or stenosis, but were abnormally situated. The cecum lay in the upper part of the abdominal cavity near the midsagittal plane, while the colon ran from this point across the left half of the upper abdominal cavity to the splenic flexure, at which point it made a right angle turn and descended normally along the left side of the peritoneal cavity to the rectum, which descended in the midline of the pelvis to the anal orifice. In addition, the whole segment of colon was suspended from the posterior abdominal wall by a thin mesentery. The liver was normally situated in the right upper quadrant and appeared unremarkable. The head of the pancreas lay within the loop of the duodenum on the left side of the peritoneal cavity and extended across the midline to the right with the tail of the pancreas lying in the vicinity of the gallbladder. No spleen was noted within the peritoneal cavity and the normal peritoneal reflections could not be traced. The foramen of Winslow was also not identified. No other abnormalities were noted within the peritoneal cavity. In the thoracic cavity, no pericardial or pleural effusions were noted, but the lungs were grossly atelectatic and appeared to be compressed by a greatly enlarged heart which weighed 190 gm. The heart was a cor biloculare with a rudimentary right ventricle, an inter-atrial septum secundum defect, an interventricular atrioventricular communis defect, an infundibular pulmonic stenosis and total anomalous pulmonary venous drainage. The pulmonary artery and aorta both arose from



FIG. 4. A lateral roentgenogram made during an upper gastrointestinal tract examination confirms the impression obtained from Figure 2 where the pylorus and the duodenum are seen directed anteriorly.

the common left ventricle with the pulmonary valve ring lying to the right of the aortic valve ring. The peripheral distribution of the vessels from the pulmonary artery and aorta was unremarkable and the ductus arteriosus was not patent. This case, therefore, demonstrates multiple congenital anomalies involving the heart and gastrointestinal tract, as well as asplenia.

DISCUSSION

The primitive gastrointestinal tract develops as a straight tube suspended in the sagittal plane by a common dorsal mesentery. It may be divided into the foregut, *i.e.*, from the mouth to the duodenojejunal junction; the midgut, *i.e.*, from the duodenojejunal junction to the midtransverse colon; and the hindgut, *i.e.*, from the midtransverse colon to the anus.

FIRST STAGE OF MIDGUT ROTATION

As the primitive intestinal tract elongates, it bulges through the umbilical orifice into the primitive umbilical cord as a temporary physiologic umbilical herniation. Failure of rotation beyond the first stage and retention of the midgut loop in the umbilical stalk at birth results in a condition known as omphalocele.

SECOND STAGE OF MIDGUT ROTATION

Nonrotation. The midgut loop is returned to the peritoneal cavity from the temporary umbilical herniation without having rotated beyond the horizontal plane it occupied at the end of the first stage.

The small bowel is entirely in the right side of the abdomen, the colon in the left. The terminal ileum crosses the midline to enter the cecum from the right. The duodenum descends entirely on the right side of the vertebral column. Internal hernias are explained as anomalies of intestinal rotation.

Paraduodenal Hernia. This is represented by imprisonment of the small bowel under the mesentery of the right colon during the process of fixation of the midgut loop after its rotation.

Reversed Rotation. Clockwise instead of counterclockwise rotation of 180° occurs during this stage. The transverse colon lies under the superior mesenteric artery and the duodenum above it.

THIRD STAGE OF MIDGUT ROTATION

This entity is characterized by descent of the cecum from the subhepatic position into the right lower quadrant and fixation of the mesentery of the cecum and ascending colon in the right flank.

An anomaly of the third stage is the subhepatic cecum. The cecum fails to elongate and descend into the right iliac fossa. If, with the cecum in this position, there is also failure of fixation of the small bowel mesentery on a broad attachment to the posterior abdominal wall, volvulus of the entire small intestine may occur. Other anomalies include retrocecal appendix and mobile cecum which we will not discuss here.

COMMENT

Omphalocele as the prime anomaly of the first stage of midgut rotation, errors of the second stage of midgut rotation giving rise to torsion of bowel segments on an abnormally persistent mesentery with bowel obstruction, and internal hernias have been adequately described in the literature. However, cases of the most primitive position of the upper gastrointestinal tract with failure of fetal rotation of the stomach before the first stage of midgut rotation (prior to descent of the gastrointestinal tube into the umbilical cord) have rarely been reported.

Caffey, in his book of Pediatric X-ray Diagnosis describes congenital microgastria and lack of fetal rotation of the stomach with failure of the greater and lesser curvature to develop. There is no differentiation into the fundus, body, antrum and pyloric canal. The tiny tubular stomach remains in the midsagittal plane and is joined from above and behind at its summit by the esophagus. The duodenum projects directly ventrad from the pyloric end of the stomach. Vomiting from birth,

hematemesis, loss of weight, and secondary anemia are the chief clinical findings.

Our case is an example of the most primitive stomach lying in the right upper quadrant in the midsagittal plane with the pylorus and duodenal bulb directed ventrad. Failure of rotation of the foregut, in addition to anomalies of second stage midgut rotation, multiple congenital cardiac anomalies and asplenia were noted.

The combination of an absent spleen and congenital heart disease, also found by other investigators,[1,8,10] is apparently more than coincidental.

SUMMARY AND CONCLUSION

A case of anomalous position of the foregut and multiple congenital heart abnormalities as well as asplenia in a two and one-half year old female is presented in which the foregut anomaly was visualized by barium opacification. The roentgen findings were confirmed on autopsy.

The position of the stomach and duodenum in this case reflects the most primitive position of the foregut. The stages of midgut rotation and other gut anomalies are briefy described.

Berta Rubinstein, M.D.
Montefiore Hospital
210th St. & Bainbridge Avenue
New York 67, New York

We are grateful for the help and advice given us by Dr. John Caffey and Dr. Harold G. Jacobson in the preparation of this manuscript.

REFERENCES

1. AGUILAR, M. J., STEPHENS, H. B., and CRANE, J. T. Syndrome of congenital absence of spleen with associated cardiovascular and gastroenteric anomalies. *Circulation*, 1956, *14*, 520–531.
2. CAFFEY, J. Pediatric X-Ray Diagnosis. Third edition. Year Book Publishers, Inc., Chicago, 1956.
3. DIDE, M. Sur un estomac d'adulte à type fetal. *Bull. coc. Anat.*, 1894, *69*, 669.
4. FINDLAY, C. W., JR., and HUMPHREYS, G. H. II. Congenital anomalies of intestinal rotation in adult. *Internat. Abstr. Surg.*, 1956, *103*, 417–438.
5. GARDNER, C. E., JR. Anomalies of intestinal rotation in gastrointestinal practice. *Am. J. Gastroenterol.*, 1955, *24*, 448–454.
6. GARDNER, C. E., JR. Surgical significance of anomalies of intestinal rotation. *Ann. Surg.*, 1950, *131*, 879–898.
7. KIESEWETTER, W. B., and SMITH, J. W. Malrotation of midgut in infancy and childhood. *Arch. Surg.*, 1958, *77*, 483–491.
8. LYONS, W. S., HANLON, D. G., HELMHOLZ, H. F., JR., DU SHANE, J. W., and EDWARDS, J. E. Cardiac Clinics, CXLVIII. Congenital cardiac disease and asplenia; report of seven cases. *Proc. Staff Mayo Clin.*, 1957, *32*, 277–286.
9. MANSON, G. Anomalies of intestinal rotation and mesenteric fixation. *J. Pediat.*, 1954, *45*, 214–233.
10. PLOWMAN, D. E. M. Congenital absence of spleen associated with cardiac abnormalities. *Brit. Med. J.*, 1957, *1*, 147–148.
11. SNYDER, W. H., JR., and CHAFFIN, L. Embryology and pathology of intestinal tract: presentation of 40 cases of malrotation. *Ann. Surg.*, 1954, *140*, 368–380.

VOL. 86, NO. 3

HYPERTROPHIC HEMANGIOENDOTHELIOMA OF INFANCY*

SUCCESSFUL MANAGEMENT OF A CASE WITH THE PATHOLOGIC AND ROENTGEN THERAPY CRITERIA

By EDWIN J. LIEBNER, M.D.
CHICAGO, ILLINOIS

THE purpose of this report is to describe the successful management of a hemangioendothelioma of muscle in a newborn infant. A review of the pertinent literature is made and certain basic principles are stressed. The textbooks on radiotherapy give little information on this entity. The pathologic diagnosis can often lead to confusion, and the surgeon must be alerted to avoid extensive mutilating procedures. It is hoped that proper direction can be given from the experience gained in this study.

The difficulties that can arise in the pathologic diagnosis, in the surgical approach, and in the necessity for prolonged fractionation of radiation dosage are well illustrated in the following case report. The mother's refusal of surgical procedures, early and again later in the course, is probably the most significant factor in the results obtained.

REPORT OF A CASE

This four day old white female was admitted to the University of Illinois Hospitals on August 10, 1956. She was born at full term, delivery was spontaneous and she weighed 7 pounds. At birth, a large tumor on the left side of the upper back and scapula extending over the left clavicle and into the left side of the neck was noted (Fig. 1, *A* and *B*). The mass felt both hard and cystic in different areas. There appeared to be a light bluish tinge to the skin but the skin itself was not involved (Fig. 2). White blood cell count was 15,000, neutrophiles 83 per cent, lymphocytes 12 per cent, and monocytes 5 per cent, with adequate platelets.

The parents were opposed to any mutilating procedure of amputation of the arm. However, they did consent to a biopsy. On August 13, 1956, under general anesthesia the patient was explored. An incision was made over the mass posteriorly for at least 10 cm. The tumor mass lay beneath the latissimus dorsi, rhomboid and trapezius muscles. Biopsy of the mass was taken at the medial border, which was adjacent to the spinous processes and transverse processes of the vertebral bodies. There was no paraspinous musculature in this area. Since the parents had denied permission for removal of the entire upper extremity and the surgeon felt that local excision could not be accomplished, only a biopsy was done.

A frozen section was reported as a highly cellular sarcoma. Subsequent study of the tissue revealed sheets of tumor cells with small oval nuclei arranged in an irregular closely interwoven pattern. In some areas the cells had larger, pale staining nuclei and indistinct cytoplasm. In other areas the cells were spindle shaped and there were numerous small vascular spaces of capillary size, some containing red blood cells. The final impression was "malignant tumor of the shoulder region, with hemangioendotheliomatous qualities" (Fig. 3).

From August 23, 1956, to September 28, 1956, the patient received the first course of irradiation via multiple small 4×5 cm. oblique portals (anterior and posterior superior and inferior, and posterior medial and lateral) from a 220 kv. machine, 1.12 mm. Cu half value layer, 50 cm. distance, with a daily dose of 75 r to a portal. Each of five portals received approximately 700 r air dose.

In the early spring of 1957, the mother reported that there was some decrease in the size of the tumor (Fig. 4). A second course of irradiation was given June 19, 1957, to July 17, 1957, through eight small 4×5 cm. oblique tangential portals. The same quality of radiation was used as in the first course and each of the portals received a total air dose of 650 r. In the ensuing six months the surgeons felt that

* From the Department of Radiology, College of Medicine, University of Illinois, Chicago, Illinois.

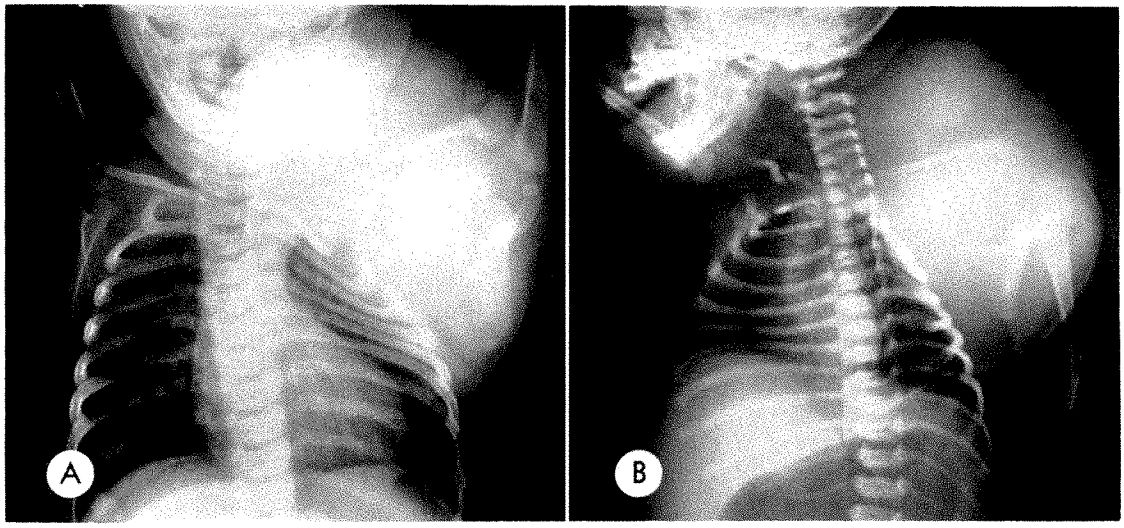


FIG. 1. (*A*) Anteroposterior and (*B*) left anterior oblique roentgenograms of the chest when the patient was nine days old. Note the large soft tissue tumor and the extent of deformity of the left upper thoracic rib cage. There is separation of the transverse processes of the lower cervical and upper thoracic vertebrae.

the lesion was now resectable, but the mother refused any surgical intervention.

In 1958, there was further slow steady reduction in the size of the mass. By this time there was no deformity of the thoracic rib cage (Fig. 5). The patient, when seen in January, 1961, revealed only the scar from the previous biopsy and a slight defect from the lack of development of normal musculature along the left superior paraspinal region, the site of previous tumor (Fig. 6).

DISCUSSION

Dargeon[1] cites a good clinicopathologic classification of lesions of vascular origin. Of the twelve categories, only two are of concern in this presentation: (a) hypertrophic hemangioma, and (b) hemangioendothelioma. His illustration of 2 cases of the hypertrophic form are exceedingly pertinent. One of them is a diffuse subcutaneous lesion of the skin itself. This grossly resembles the case being presented. However, all of his tabulated hypertrophic cases have associated thrombocytopenic purpura. Dargeon also stated that in the therapy of this lesion, roentgen therapy is frequently unsatisfactory and surgery may be required. Southard *et al.*[3] reported a twenty-one day old infant who had a capillary hemangioma of the left leg and purpura. The lesion received 3,360 r in twenty days, and three weeks later had completely disappeared. Three months later, a second course of 2,400 r was given for an extension. Contracture and radiation fibrosis was reported.



FIG. 2. Clinical appearance before irradiation, when the patient was seventeen days old. Note the lack of any skin involvement.

Watson and McCarthy[6] have written on a hypertrophic or angioblastic type of hemangioma. They stated that this appears chiefly in the skin as a solid elevated purplish red lesion. They further commented on the histologic features as being masses of cells with a minimum of patent vessels. The pure type can be mistaken for hemangiosarcoma and is frequently recur-

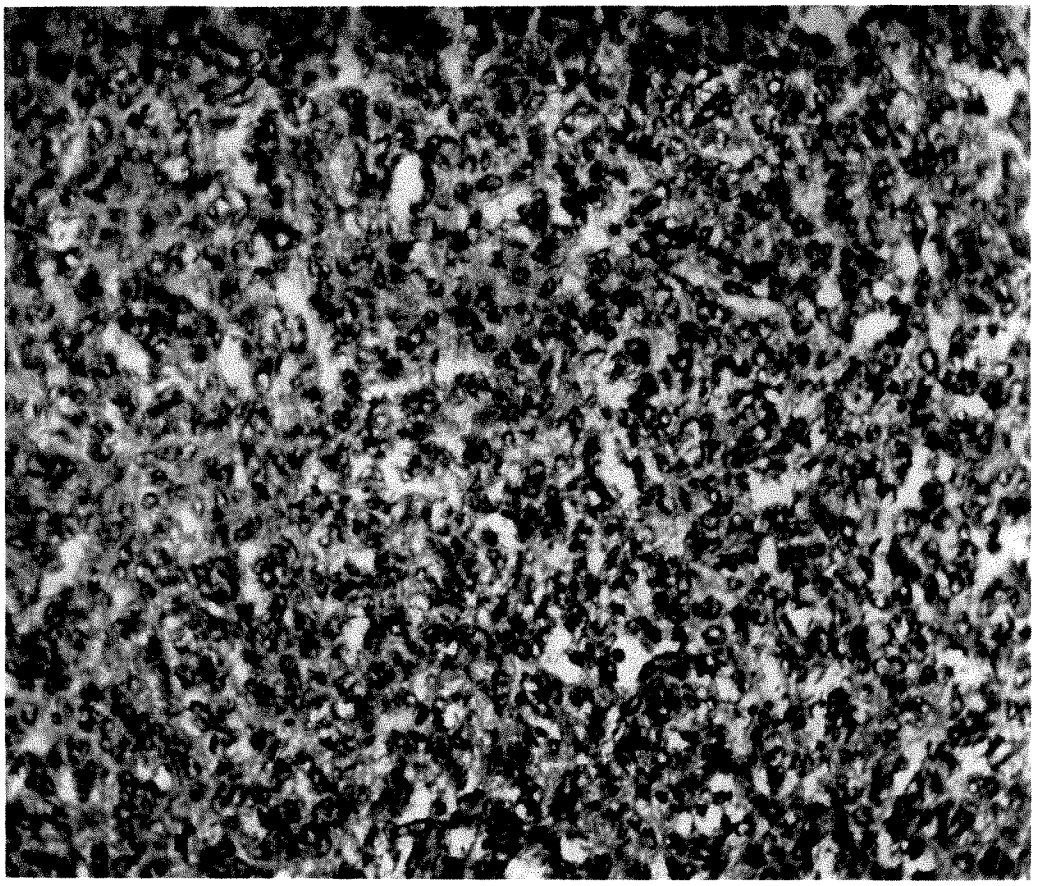


FIG. 3. Microscopic section of the biopsy specimen (×360). The tumor was reported as malignant with hemangioendotheliomatous characteristics.

rent after surgery, which indicates its local aggressiveness.

Stout[4,5] has contributed greatly to the understanding of the pathology of malignant tumors of vascular origin. All are characterized by the formation of vascular channels, but with different cells playing a dominant role in tumor growth. If cells of the inner lining are predominant, we have (a) the hemangioendothelioma; if the muscular layer of the vessel is active, then (b) a leiomyosarcoma results; and, finally, if the tumor originates in cells of the outer surface sheath of the vessel, it is (c) the hemangiopericytoma.

The reticulum stain is of great value in differentiating between hemangioendothelioma and hemangiopericytoma. The silver reticulum stain of the histologic slide will show that all the cells are inside of the reticulum sheaths and are, therefore, endothelia, while the reverse is true for the pericytoma. Besides this, Stout stresses that in a hemangioendothelioma there is formation of atypical endothelial cells in greater numbers than are required to line the vessels with a simple endothelial membrane, formation of vascular channels with a delicate framework of reticulum fibers and a marked tendency for their lumens to anastomose. The pathologic material in the present case was studied by the author with the use of the reticulum stain and it shows the features of a hemangioendothelioma as described by Stout (Fig. 7).

In regard to the malignant qualities of this tumor and the prognosis, several salient features should be emphasized. McCarthy and Pack[2] and Stout[4] have stressed that there is one variety of hemangioendo-



FIG. 4. Left shoulder and back of the infant at ten and one-half months of age, nine months after the initial irradiation and prior to the second course of therapy. Note the areas of tumor regression where the effect of irradiation from the tangential and oblique portals was greatest.

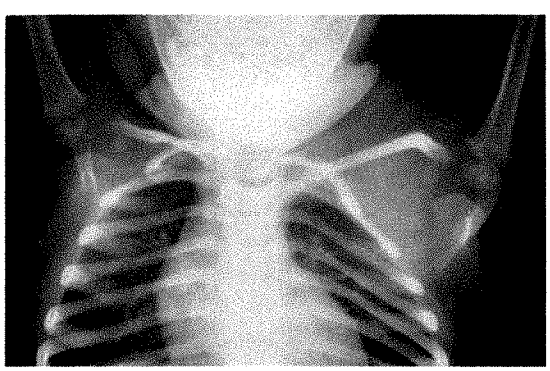


FIG. 5. Anteroposterior view of the chest at the age of one year and five months, or six months after the second course of irradiation. Note the marked decrease of soft tissue tumor, early calcification, and improvement of the thoracic rib deformity. (Compare with Figure 1.)



FIG. 6. The upper back four and one-half years after irradiation. Note the long scar resulting from the incision for the biopsy, and the atrophy or absence of muscles in this area caused by the congenital replacement of any normal musculature by the tumor. The darkening of the skin is exaggerated by a photographic shadow, but irradiation changes are minimal.

thelioma which is seen in infancy or early childhood and which has a rather benign course. McCarthy and Pack caution against the acceptance of the ambiguous term, "hemangioendothelioma," in children. They further state that hemangioendothelioma connotes a highly malignant sarcoma (as our pathologist suggested) and often the surgeon desires to do an amputation, as in our case.

The radiation therapy of hemangioendothelioma of infancy can present difficulties if its proper pathologic identity is not recognized. Many radiotherapists are acquainted with the method and techniques of treating hemangiomas in infants. The chief criteria are early treatment with small doses fractionated over a long interval to supplement or augment the tendency to natural regression or spontaneous remission. The author believes that hemangioendothelioma of infancy, especially the hypertrophic type, should be treated similarly. There is considerable danger if the pathologic state is not recognized and it is interpreted as a form of malignant sarcoma. The surgeon will always do radical surgery and the radiotherapist may be tempted to give a large cancerocidal dose in the usual short interval of four to five

weeks. There will be resulting skin damage and the normal structures will be prevented from developing properly. Since the child will survive long after the radiation has been given, there will be greater dangers of even more serious complications, such as cancer developing in the skin and bone in the long latent period.

If the lesion is large, as it was in our case, it will be advisable to avoid the use of large direct fields. Small multiple tangential fields to avoid the deeper structures are most appropriate. It is necessary to deliver radiation only to a major part of the lesion, since total regression will occur eventually.

From the factors used in this case, a tumor depth dosage can be derived. However, it is difficult to calculate the depth dose accurately since the radiation was directed obliquely and tangentially through multiple small fields to avoid a large dose to the underlying structures. Nevertheless, for theoretical purposes, at a 5 cm. depth in the lesion, and assuming that all the five portals contributed, we may conclude that approximately 2,000 r was delivered to the maximum point and at a 10 cm. depth, a dose of 1,000 r. This was over a period of five weeks. The lesion was observed to have regressed moderately over a ten month period and then a nearly similar course of irradiation was given as described in the case report. The figures cited are maximum values, but in reality they probably were considerably lower. In retrospect, it is probable that a lower dosage would have been utilized if we had better understood the pathologic characteristics and anticipated the degree of radiation response.

The result from the radiation treatment has been most gratifying. There has been very little skin change, and the thoracic rib

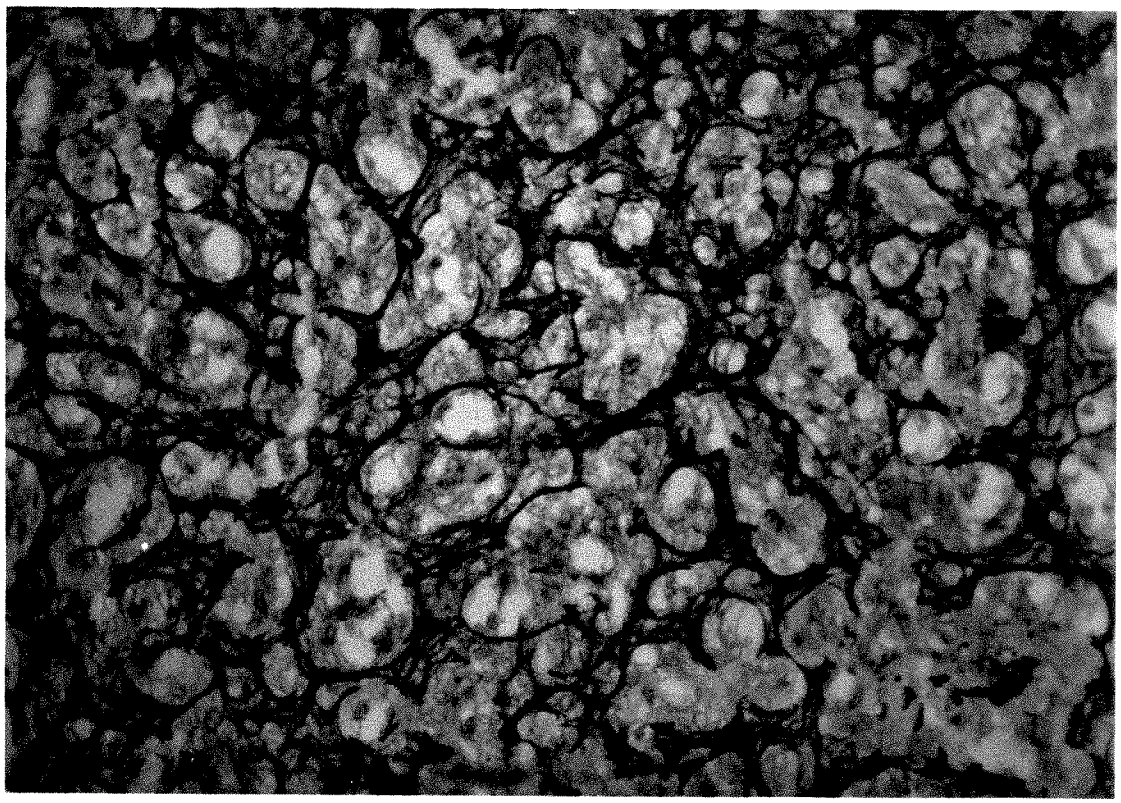


FIG. 7. Microscopic section with a reticulum stain (×315), showing the chracteristic histology of a hemangioendothelioma (Stout). The section demonstrates the great number of endothelial cells lining the lumen of anastomosing vessels.

and vertebral development have been normal (compare Figure 1*A* and Figure 8). Since the tumor originated in or congenitally replaced the left upper spinal musculature, there is a slight atrophy or lack of development in this region following the tumor regression (Fig. 6). Roentgen study shows a normal chest cage (Fig. 8). A moderate amount of calcification has developed in the area of the former tumor (Fig. 9); it is a monument to the history, course, and treatment of this disease. The child is nearing the completion of her fifth year after therapy and there are no indications of any recurrence.

CONCLUSIONS

Proper recognition of this vascular lesion in infancy and early treatment are believed to be essential if radiation therapy is to be used. If we follow Stout's pathologic criteria and also consider the lack of any purpura, the benign course of a hypertrophic lesion in infancy, and the protracted radiation response, the lesion in the infant studied is best classified as a hypertrophic hemangioendothelioma of infancy.

The infant was seventeen days old when therapy was instituted. Radiation dosage should be in a low to moderate range, depending upon the extent and bulk of the lesion. The irradiation should be fractionated over a long period to observe the response and to take advantage of any natural or spontaneous regression that may occur.

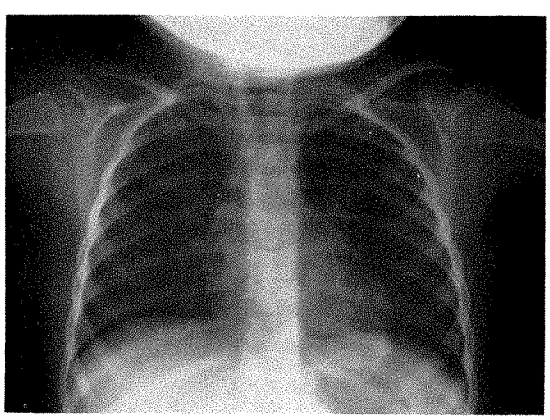


FIG. 8. Anteroposterior view of the chest four years after irradiation. Note the good development of the left upper chest and the calcification in the left supraclavicular region.

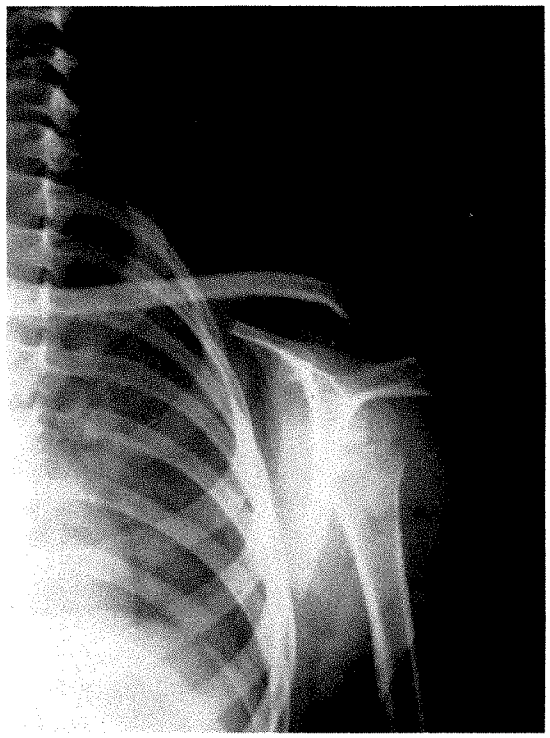


FIG. 9. Left oblique view of the shoulder demonstrating calcification at the former site of the tumor.

SUMMARY

1. A case of hypertrophic hemangioendothelioma in a seventeen day old infant is reported. The lesion originated in the left upper thoracic paraspinal musculature.

2. The pathologic and clinical criteria are given for the proper identification of the lesion.

3. Because of the frequently benign nature of this lesion, extensive surgical procedures should be avoided.

4. Radiation dosage should be in the lower range and the course should be protracted to augment a possible natural tendency to regression.

5. The reported child has developed well with no radiation sequelae. The site of the

former tumor shows calcification five years later.

840 South Wood Street
Chicago 12, Illinois

Dr. Arthur S. J. Petersen was most helpful in suggesting the use of multiple small fields.

REFERENCES

1. Dargeon, H. W. Tumors of Childhood; A Clinical Treatise. Paul B. Hoeber, Inc., New York, 1960, pp. 358–384.
2. McCarthy, W. D., and Pack, G. T. Malignant blood vessel tumors; report of 56 cases of angiosarcoma and Kaposi's sarcoma. *Surg., Gynec. & Obst.*, 1950, *91*, 465–482.
3. Southard, S. C., DeSanctis, A. G., and Waldron, R. J. Hemangioma associated with thrombocytopenic purpura; report of case and review of literature. *J. Pediat.*, 1951, *38*, 732–737.
4. Stout, A. P. Hemangioendothelioma; tumor of blood vessels featuring vascular endothelial cells. *Ann. Surg.*, 1943, *118*, 445–464.
5. Stout, A. P. Tumors of soft tissue. Atlas of Tumor Pathology, Fasc. 5, Section II. Armed Forces Inst. of Path., Washington, D. C., 1953.
6. Watson, W. L., and McCarthy, W. D. Blood and lymph vessel tumors; report of 1,056 cases. *Surg., Gynec. & Obst.*, 1940, *71*, 569–588.

A SIMPLE METHOD OF REDUCING THE FLUOROSCOPIC DOSE RATE

By EDWIN L. LAME, M.D.
PHILADELPHIA, PENNSYLVANIA

TO IMPLEMENT some of the recent thought on fluoroscopic safety, we started a plan three years ago that has proved most satisfactory and seems easily applicable for all users of modern machines. The roentgen dose rate can be regulated at will for each case and can be so reduced that babies and pregnant women may be examined with hitherto unaccustomed low dosage.

Since, even today, there is evidence that some fluoroscopes are operated by radiologists at high r rates (up to 46 r per minute at table top[4]), it may be useful to review a method that is so simple.

The best fluoroscopic techniques should be in operation in the children's hospitals. Our analysis of current practice in thirteen children's hospitals shows that in orthodox fluoroscopy the kv. peak ranges from 40 to 95, the ma. from 1 to 4, and the r/min. from 1 to 7.5. The average usual kv. peak is 73, the average ma. is 2.5, and the average r/min. is 3.6 (Table 1). This is an enviable record for those using 1 r/min. and we wish to publicize such excellent habits for the information of all radiologists. It would be desirable to reduce the



FIG. 1. Fluoroscopic ma. meter large enough for easy reading and setting.

TABLE I

FLUOROSCOPIC FACTORS USED IN 13 CHILDREN'S HOSPITALS IN THE UNITED STATES

Hospital	Usual			Highest			Lowest		
	kv. peak	ma.	r/min.	kv. peak	ma.	r/min.	kv. peak	ma.	r/min.
1	70	2	1.9	90	3	2.8	60	1	1
2	85	1.5	1.75	95	3	3.2	65	1	1.1
3	70	2.4	1.9	90	2.4	3.1	55	2.4	1.1
4	80	4	3.6	90	4	4.6	65	4	2.1
5	70	2	4.5	80	3	7.5	60	1	2.2
6	85	3	5	95	3	5	55	3	5
7	95	2	2.25	95	3	3.5			
8	60	2	1.66	70	2		50	2	
9	65	3	4	75	3	5		3	
10	70	3	9						
11	55	2		65	2		40	2	
12	74	3		78	3		68	3	
13	80	3		85	3		75	3	

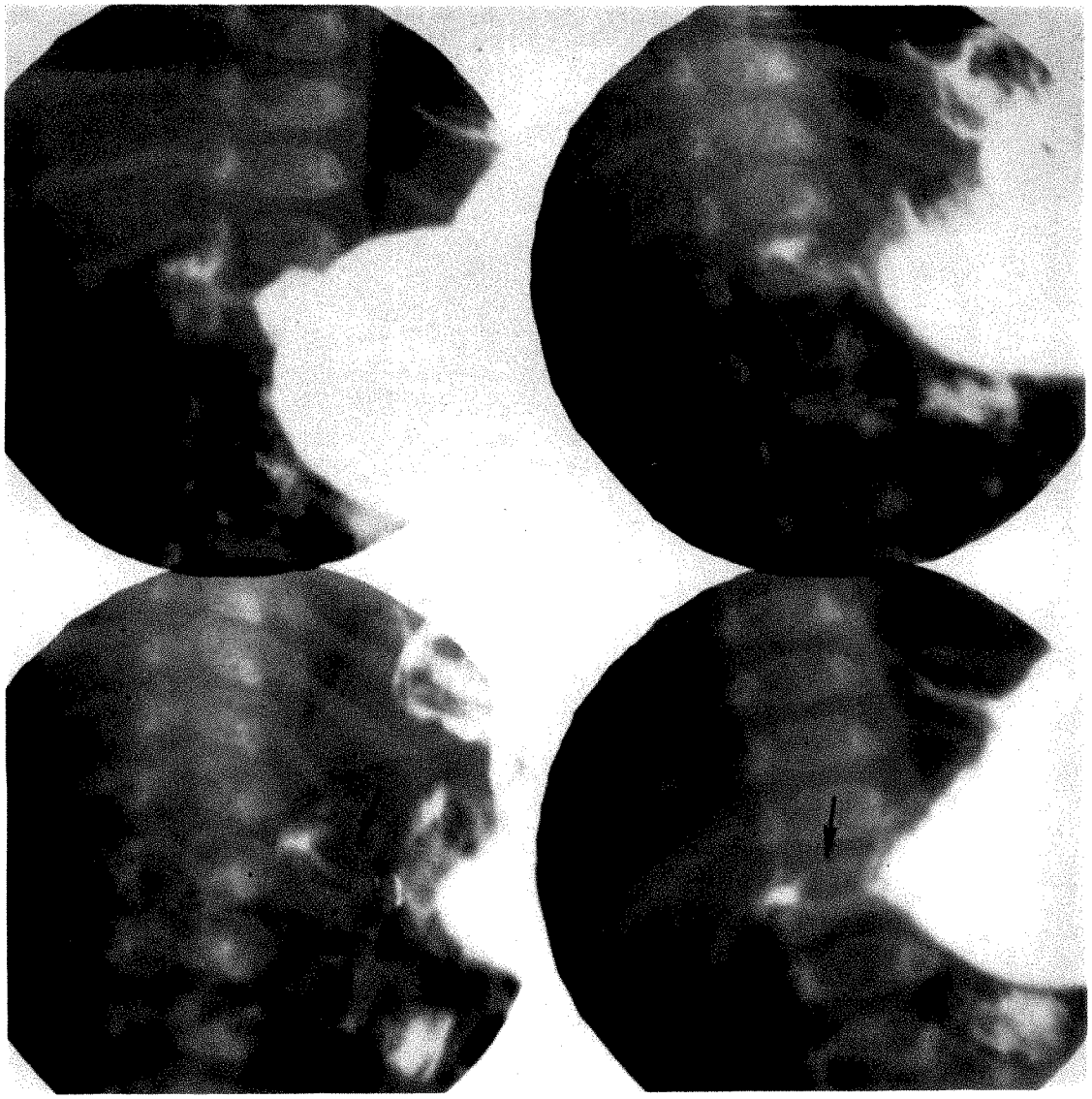


FIG. 2. Roentgenograms made on 8×10 inch film carrying 4 exposures. Diameter of the aperture was 8 cm.

higher rates for children and, further, to offer adults a similarly low exposure approximating the children's dosage. This seems realized in our recent program.

RECOMMENDED METHOD

Most new control panels have a hand regulator of fluoroscopic milliamperage, but manufacturers will equip old panels if requested. This hand regulator is locked at a maximum of 3 ma., forcing the operator to use a higher kv. peak and promptly reducing r output.

Recently, a wide scale milliammeter has been purchased (Fig. 1). It is easy to adjust and read and is much preferred by the physicist, technician and radiologist.

The most gratifying results are in the ability to fluoroscope babies and children or pregnant women with a very low total dose. The calibrating physicist measures the dose at required factors and these dose rates are posted in the technician's booth at each machine. At the start of each fluoroscopic examination the technician asks the radiologist his needs for voltage

and current. Sample readings from our 3 machines are as follows:

kv. peak	ma.	r per minute		
60	0.5	0.95	1.0	0.7
60	1	1.53	1.8	1.2
70	1	2.14	2.6	1.6
85	1	3.4	3.5	2.2
85	2	4.76	6.4	3.5
90	2	5.8	7.7	5.1
90	3	6.7	9.8	7.0

Reference to the operating factors above makes it evident that an infant can be examined at 60 kv. peak and 0.5 ma. for an r rate of 0.7 to 1.0 per minute. Our barium fluoroscopy for an infant usually is limited to 1 minute and hence a dose of about 1 r is maintained.

Further reduction of total body dose for babies is effected by using the 8 cm. diameter 4 on 1 spot filmer as a fixed aperture during all of the fluoroscopy and most of the roentgenography (Fig. 2).

Correspondingly, thin adult chests can be examined at dose rates of 1.6 to 2.6 r per minute (70 kv. peak-1 ma.). Adult barium examinations are done at 85 kv. peak and 1 ma. for thin patients, 85 to 90 kv. peak and 2 or 3 ma. for heavier patients (dose rate of 2.2 to 9.8 r/min.).

Comparison with image amplifier dose rates commonly used is most favorable. Most users of the amplifiers do not accomodate their eyes, so that their rates of exposure are the same as ours for children and light-weight adults, and sometimes higher for babies.

SUMMARY

A simple and very inexpensive plan is described of reducing roentgen dose rate in fluoroscopy. It has provided the author with the means for examining delicate tissues that otherwise would be rejected for fluoroscopy or overdosed by current standards of safety.

It is suggested that this method is worthy of wider trial in view of the continued prevalence of more robust fluoroscopic examination.

Presbyterian Hospital
Philadelphia 4, Pennsylvania

REFERENCES

1. BILES, E. Personal communication.
2. FEIST, J. H. Personal communication.
3. GIRDANY, B. Personal communication.
4. GORSON, R. O., LIEBERMAN, J., and GREEN, M. Limited survey of radiation exposure from medical fluoroscopes. *Radiology*, 1959, *73*, 898–910.
5. HOPE, J. Personal communication.
6. HOWARD, W. H. Personal communication.
7. KING, G. G. Personal communication.
8. LATTMAN, I. Personal communication.
9. MABRY, E. Personal communication.
10. PETERSON, C. A. Personal communication.
11. PHILLIPS, L. A. Personal communication.
12. PIRKEY, E. L. Personal communication.
13. SCHREIBER, R. R. Personal communication.
14. TROUT, E. D., KELLEY, J. P., and LUCAS, A. C. Influence of cable length on dose rate and half-value layer in diagnostic x-ray procedures. *Radiology*, 1960, *74*, 255–264.
15. WHITE, H. Personal communication.

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Published by: CHARLES C THOMAS, PUBLISHER, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued monthly. Annual subscription: United States, Mexico, Cuba, Central and South America, $15.00; Canada, $16.00; other countries, $17.00. Current single numbers, $1.50. Advertising rates submitted on application. Editorial office, 110 Professional Building, Detroit 1, Michigan, Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois. Information of interest to authors and readers will be found on page ii.

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Forty-fourth Annual Meeting: Waldorf-Astoria Hotel, New York, N. Y., April 2–4, 1962.

* *Ex officio.*

EDITORIAL

THE THYMUS GLAND IN CHILDREN AND THE ROENTGENOLOGIST

PROBLEMS related to the thymus gland in children continue to provide grounds for speculation and controversy. The general roentgenologist does not have as frequent an opportunity to familiarize himself with thymic disease as does the pediatric roentgenologist. Nevertheless, he does encounter recurring experience with two basic problems. The first is a diagnostic question: Is the thymus gland enlarged? The second is a therapeutic problem: Should the thymus be treated by radiation? A historical survey of the roentgenologist's association with the thymus gland helps to elucidate our present role.

Medical knowledge of the presence of the thymus gland probably dates from antiquity. In an extensive review, Marine[1] credited Felix Plater with introducing in 1614 the concept of sudden death in children due to an enlarged thymus gland. In 1889 Paltauf[2] focussed new interest on the thymus gland by introducing the idea of a constitutional disease, status thymicolymphaticus, consisting of hyperplasia of the lymphoid structures including the thymus, arterial hypoplasia, chlorosis, and a tendency to unexplained sudden death.

Only eight years after Röntgen's discovery of the x-ray, Heineke[3] described the effects of the x-ray on lymphoid tissue in experimental animals and suggested that such therapy might have value in human beings with abnormally large thymus glands. Friedlander[4] put this hypothesis to trial in 1905, when he treated an infant with a respiratory ailment thought to be due to thymic stridor and status lymphaticus. By this time it was widely believed that these patients responded poorly to anesthetic drugs and suffered a high rate of sudden, unexpected death. That radiation was increasingly accepted as a useful means of controlling the enlarged thymus and its feared consequences is readily attested by a review of early volumes of the AMERICAN JOURNAL of ROENTGENOLOGY.

In a report of 41 cases in 1920, Heublein[5] gave the reasons why radium therapy was considered even more useful than roentgen therapy. Grier[6] agreed that radium was easier and safer to use; his report is notable because it emphasized also the responsibility of the roentgenologist in the diagnosis of the enlarged thymus. In a roentgenographic study, he stressed the importance of the phase of respiration, the increased size of the thymic shadow when the child cries, and the tendency of the shadow to be bilateral, relatively symmetric, and wider inferiorly. Writing at a still later date, Köhler[7] described graphically the difficulty encountered by earlier diagnosticians: "In the first decade after Röntgen, enlargement of the thymus was far too frequently diagnosed, the striking breadth of the upper part of the central shadow being due to the tube being brought too near, in order to take a more rapid picture of small unruly children. . . ."

[1] MARINE, D. Status lymphaticus. *Arch. Path.*, 1928, *5*, 661–662.

[2] PALTAUF, A. Cited by Friedlander.[4]

[3] HEINEKE, H. Ueber die Einwirkung der Röntgenstrahlen auf innere Organe. *München. med. Wchnschr.*, 1904, *51*, 785–786.

[4] FRIEDLANDER, A. Status lymphaticus and enlargement of the thymus. With report of a case successfully treated by the x-ray. *Arch. Pediat.*, 1907, *24*, 490–501.

[5] HEUBLEIN, A. C. Radiation treatment of enlarged thymus glands in infants. AM. J. ROENTGENOL. & RAD. THERAPY, 1920, *7*, 191–195.

[6] Grier, G. W. The diagnosis and treatment of enlarged thymus. AM. J. ROENTGENOL. & RAD. THERAPY, 1924, *11*, 141–146.

[7] KÖHLER, A. Roentgenology: The Borderlands of the Normal and Early Pathological in the Skiagram. Second English Edition. Wm. Wood & Co., Baltimore, 1935, p. 477.

The popularity of radiation therapy met some resistance. In 1927 Greenwood and Woods[8] described the whole concept of status thymicolymphaticus "as a good example of the growth of medical mythology." Opposing thymic irradiation, Kennedy and New[9] reported that chronic stridor and other symptoms commonly attributed to an enlarged thymus were usually due to causes unrelated to the thymus. In general, however, roentgenologists and other physicians diagnosed and treated an increasing number of large thymus glands. The peak of this fad was reached in the late 1930's, when many children were treated whether they had symptoms or not. In the 1940's, doubts began to develop about the importance of status thymicolymphaticus, but supposedly symptomatic thymus glands were still subjected to radiation therapy with few qualms about the danger involved.

With the advent of the atomic era and the cataclysmic events at Hiroshima in 1945, the uses of radiation therapy underwent thoughtful reappraisal. Coming under particular scrutiny was its use in children, women in the child-bearing age, and persons having benign diseases. In 1949, Scharnagel and Pack[10] reported the appearance of hundreds of basal cell epitheliomas in the skin of a five year old boy whose enlarged thymus had been irradiated shortly after birth. In 1950, Duffy and Fitzgerald,[11] and, five years later, Clark[12] recorded cases of children with thyroidal cancer, which they associated in many instances with irradiation that had been given during infancy to the thymus or to other benign conditions of the upper trunk. In this same period Dameshek[13] and Simpson and coworkers[14] were pointing out the possibility that leukemia might be associated with irradiation of the thymus in children.

Medical contributions such as these certainly reduce the enthusiasm for irradiation of the thymus gland. During the last decade, also, in some nonradiologic quarters near-hysteria has existed about the observed or projected hazards of radiation damage. Insofar as these developments lead to fuller assessment of our knowledge and to wiser use of radiation therapy, they must be accepted as good. This greater awareness of certain disadvantages, however, should not blind us to the usefulness of radiation therapy, judiciously employed. Recent conscientious studies of large series of patients[15,16] indicate suggestive evidence that thymic irradiation in children may potentially be harmful. A still larger study[17] indicates that it may be a contributing factor to development of cancer of the thyroid. In a similar report by Conti and others,[18] which did not disclose a relationship in the development of either leukemia or thyroidal neoplasm, very small ports (4×4 cm.) were used directly over the thymus. This report states that careful, limited irradiation of the thymus may be undertaken without undue risk. The roentgenologist may observe in these reports an interesting telltale—that the departments from which the studies originate have largely abandoned use of irradiation for benign thymic enlargement.

The answer to our first question, then,

[8] Greenwood, M., and Woods, H. M. "Status thymicolymphaticus" considered in light of recent work on thymus. *J. Hyg.*, 1927, *26*, 305–326.

[9] Kennedy, R. L. J., and New, G. B. Chronic stridor in childhood sometimes erroneously attributed to enlargement of the thymus. *J.A.M.A.*, 1931, *96*, 1286–1289.

[10] Scharnagel, I. M., and Pack, G. T. Multiple basal cell epitheliomas in a five year old child. *Am. J. Dis. Child.*, 1949, *77*, 647–651.

[11] Duffy, B. J., Jr., and Fitzgerald, P. J. Thyroid cancer in childhood and adolescence. Report on 28 cases. *Cancer*, 1950, *3*, 1018–1032.

[12] Clark, D. E. Association of irradiation with cancer of the thyroid in children and adolescents. *J.A.M.A.*, 1955, *159*, 1007–1009.

[13] Dameshek, W. The outlook for the eventual control of leukemia. *New England J. Med.*, 1954, *250*, 131–139.

[14] Simpson, C. L., Hempelmann, L. H., and Fuller, L. M. Neoplasia in children treated with x-rays in infancy for thymic enlargement. *Radiology*, 1955, *64*, 840–845.

[15] Snegireff, L. S. The elusiveness of neoplasia following roentgen therapy for thymic enlargement in children. *Radiology*, 1959, *72*, 508–517.

[16] Latourette, H. B., and Hodges, F. J. Incidence of neoplasia after irradiation of thymic region. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 667–677.

[17] Saenger, E. L., Silverman, F. N., Sterling, T. D., and Turner, M. E. Neoplasia following therapeutic irradiation for benign conditions in childhood. *Radiology*, 1960, *74*, 889–904.

[18] Conti, E. A., Patton, G. D., Conti, J. E., and Hempelmann, L. H. Present health of children given x-ray treatment to the anterior mediastinum in infancy. *Radiology*, 1960, *74*, 386–391.

is: a prominent thymus may be discovered, but its significance is doubtful. The roentgenologist must diagnose its presence and perhaps follow its involutional course in children, but should not produce undue alarm in reporting its size or supposed effect upon the trachea.

In answering the question about therapy, the roentgenologist must almost certainly accept the idea that radiation therapy for an enlarged thymus is rarely advisable. This change in emphasis is as much a product of modern knowledge about normal variations of the gland, the infrequency of pressure symptoms, and the vanishing concept of status lymphaticus as it is of concern about the possible association of subsequent thyroidal or lymphoid cancer. However, if a large thymus is obstructing respiration in a child in whom complete pediatric and otolaryngologic appraisal reveals no other cause, use of *small* doses of roentgen therapy to a *small* portal below a carefully shielded thyroid gland is justified.

Based on an appraisal of general statistics indicating a continued low incidence of thyroidal cancer in children, and a long, personal experience in irradiation of children, Garland[19] believes that "An etiological connection between low-dose neck irradiation and subsequent thyroid cancer is not regarded as established."

SEYMOUR FISKE OCHSNER, M.D.

Ochsner Clinic
Prytania and Aline Streets
New Orleans 15, Louisiana

[19] GARLAND, L. HENRY. Cancer of the thyroid and previous irradiation. *Surg., Gyn. & Obst.*, 1961, *112*, 564–566.

NEWS ITEMS

DR. OTTO GLASSER HONORED

To Otto Glasser, Ph.D., teacher, leader and friend to the radiologist of Ohio, the members of the Ohio State Radiological Society presented a scroll in recognition of his many contributions to Radiology. Also, in appreciation of these and his many other attainments as author and investigator in the field of biophysics, the Ohio State Radiological Society on May 20, 1961 conferred upon him an Honorary Membership.

THE ASSOCIATION OF UNIVERSITY RADIOLOGISTS

Annual Memorial Medal Award Competition

The Association of University Radiologists has recently established an annual Memorial Medal Award in honor of deceased members, to be awarded to the resident in radiology writing the best original paper on some aspect of radiology during the period of competition. The paper must be the work of a single author, the resident. Manuscripts to be considered for the 1962 competition must be received by March 1, 1962. Inquiries should be addressed and papers submitted to the Secretary, Herbert M. Stauffer, M.D., Department of Radiology, Temple University Medical Center, Philadelphia 40, Pennsylvania.

The 1961 Memorial Medal Award was won by Dr. Burton Seife, Resident, Yale University, for his paper, "Radioactive Inert Indicator Method for Intestinal Absorption Utilizing Differential Counting," presented at the recent meeting of the Association at Stanford University Medical Center, Palo Alto, California.

The 1962 annual meeting will be held at the University of Indiana in Indianapolis, May 19 and 20.

JAMES PICKER FOUNDATION

Awards for 1961–1962

On behalf of the James Picker Foundation, the National Academy of Sciences-National Research Council announces the award of twenty-five grants and fellowships in the field of radiology and nuclear medicine for 1961–1962. These awards, totaling approximately $150,000, were recently approved by the Foundation on recommendation of the Committee on Radiology of the Academy-Research Council. With the advice and cooperation of the Committee, the Foundation inaugurated its research grants and fellowships in 1950, with the objective of fostering research in, and broadening the scope of radiology. During the past year, the program was expanded to include a new series of Advanced Fellowships in Academic Radiology, in recognition of the increasing interest of the Foundation in the advancement of medical education. The first three of these Fellowships, designed to assist young radiologists who are preparing for careers as medical school teachers, have been awarded for 1961–1962.

At the same time, the National Research Council of Canada announces on behalf of the Foundation the award of two Research Grants for the coming academic year. Since 1958, the Canadian National Research Council has served as scientific adviser to the James Picker Foundation with respect to its Canadian program.

Including the awards for the coming year, during the eleven year period research grants have been made through the U. S. Academy-Research Council for support of seventy-one investigations throughout the world. Twenty-five universities and nine hospitals and research laboratories within the Unites States have received grants. Investigators in four Canadian institutions were awarded support before the Canadian National Research Council assumed responsibility for the Foundation's Canadian program in 1958. Grants have been made to ten institutions in seven countries abroad. Nine Scholars in Radiological Research have been supported in nine universities in this country.

Research Fellowships have been awarded to twenty-six young scientists to enable them to prepare for investigative careers in the field of radiology. Fifteen of these fellows were citizens of countries other than the United States, and eleven of the latter studied in this country or Canada. Three of the Americans have carried on their research in England and Sweden.

The Canadian National Research Council since 1958 has administered ten Research Grants in Canadian institutions, and has recommended the award of one Research Fellowship in Canada.

JAMES PICKER FOUNDATION

Application for Awards for 1962–1963

On behalf of the James Picker Foundation, the National Academy of Sciences-National Research Council is accepting applications for fellowships and grants in the field of radiology and nuclear medicine for the year 1962–1963. Awards are made in four categories:

The Advanced Fellowships in Academic Radiology. The purpose is to prepare the fellow to meet in full the intellectual demands of an academic position in radiology. Preference will be given to applicants under thirty-four years of age. Completion of clinical training in radiology ordinarily will be a prerequisite. The candidate should be prepared to devote a minimum of two years to course work in the basic sciences and to the application of the techniques and methods of these basic disciplines to radiological research. In arranging the details of his program, the prospective fellow will have opportunity for consultation with members of the Academy-Research Council's Committee on Radiology. Applications will be accepted on nomination by a clinical adviser, who should be a member of the staff of a department of radiology in a medical school.

Postdoctoral Research Fellowships are open to recent graduates who desire investigative experience and training in radiology or nuclear medicine. Applicants must hold the M.D., Ph.D. or Sc.D. degree or the equivalent. Awards are for a period of one year, but requests for renewal will be considered.

Grants for Scholars are offered to assist medical schools in supporting and developing junior staff members in radiology. This is a transitional form of support, designed to bridge the gap between the completion of research fellowship training and the period when the young scientist has thoroughly demonstrated his competence as an independent investigator. The application is submitted by the institution on behalf of the prospective Scholar. If the request is approved, a grant of $6,000 per year will be made directly to the institution as a contribution toward the Scholar's support or his research, or both. Initial grants are made for two years, but renewal for a third year may be recommended.

Grants-in-Aid of Research are designed to encourage investigations in radiology and nuclear medicine offering promise of improvement in methods of diagnosis or treatment of disease. Radiological studies involving the use of radioisotopes fall within the scope of this program. Research grants are awarded to institutions, rather than to individuals. Initial awards may be made for two years.

Applications for the fiscal year 1962–1963 should be submitted by October 1, 1961. Support is not restricted to citizens of the United States or to institutions within this country. Application blanks and further details may be obtained from the *Committee on Radiology, National Academy of Sciences-National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.*

The Medical Research Council of Canada serves as scientific adviser to the James Picker Foundation with respect to its Canadian program. If the proposed studies are to be carried out in Canada, requests for information and application forms should therefore be directed to the Secretary, Medical Research Council, National Research Building, 100 Sussex Drive, Ottawa 2, Canada.

SPECIAL PROGRAM FOR RESEARCH GRANTS IN RADIOLOGICAL HEALTH

A greatly expanded program for research grants in the field of radiological health has been developed by the U. S. Department of Health, Education, and Welfare, Public Health Service, Division of Radiological Health. These grants are offered to support research by individuals, universities, hospitals, laboratories, and other public or private institutions in the assessment and control of manmade and natural radiation exposures to the individual, no matter how the separate components may originate. The knowledge and skills of many professional disciplines and specialties—physicians, engineers, physicists, chemists, educators, statisticians among them—are needed to find answers to the many challenging questions in radiological health.

Basic studies relating to critical body organs and systems, preferred metabolic pathways for specific radioactive contaminants and an understanding of the radiosensitizing and modifying effects of various materials are encouraged. Broad epidemiological studies aimed at a scientific evaluation of the long term effects such as aging, congenital malformations, genetic effects, behavioral patterns, and cancer induction are also of primary concern. Field studies of the movement of radioactive contaminants in biota and human food chains are of special interest, since the physical environment may be greatly altered by biological activity, as for example, the concentration of waterborne radionuclides in microorganisms and fish. Purely physical studies, such as chemical mechanisms in radiation chemistry, the design of equipment and the development of techniques to accurately assess or reduce the population dosage are mandatory for a successful research program.

For information and/or application forms please contact: Dr. Paul F. Hahn, Chief, Office of Extramural Grants, Division of Radiological Health, U. S. Public Health Service, Washington 25, D. C.

OPERATIVE RADIUM THERAPY DAY AT QUEENS HOSPITAL CENTER

An Operative Radium Therapy Day is to be given at the Queens Hospital Center by the Radiation Medicine Department on Wednesday, October 18, 1961.

The program will include the use of interstitial radiation in the oral cavity; radical insertion of radium in the neck; various applicators in carcinoma of the cervix; the Heyman applicators in carcinoma of the endometrium; radium needles in the parametria; and radon, iridium-thread, and cobalt interstitial therapy. Clinical demonstration will depend on availability of patients.

The enrollment is limited and the registration fee is $10.00.

Please apply to: Dr. Philip J. Kahan, Supervising Medical Superintendent, Queens Hospital Center, 82–68 164th Street, Jamaica 32, N. Y.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

STRAHLENBIOLOGIE, STRAHLENTHERAPIE, NUKLEARMEDIZIN UND KREBSFORSCHUNG; ERGEBNISSE 1952–1958. Edited by H. R. Schinz; H. Holthusen; H. Langendorff; B. Rajewsky; and G. Schubert. Cloth. Price, $65.50. Pp. 998, with numerous illustrations and tables. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1959. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

German medical literature has demonstrated the successful use of "Ergebnisse" for a long time, *i.e.*, a collection of chapters written by different authors and pertaining to different fields of medicine. This volume is the first of its kind in the field of radiation biology, radiation therapy, nuclear medicine and cancer research. It comprises 16 different chapters, varying in length from 12 to 90 pages. There are purely scientific chapters, such as those dealing with the radiation genetics of bacteriophages, of micro-organisms, of Drosophila, and of mammals. Other chapters in the field of radiation physics are concerned with problems of dosage, and the physics and technique of megavolt radiation. The greater part of the book deals with practical aspects of radiation therapy, such as experience with the 31 megavolt betatron and intravesical radiocobalt therapy in Zurich, radiation therapy of bronchogenic carcinoma, a critical review of therapy with radioactive isotopes, clinical diagnosis of diseases of the thyroid by means of radioiodine, and others. The authors contributing to this book are working in Switzerland and Germany.

Every chapter has a very extensive bibliography for the years 1952–1956 which should be of considerable assistance to anyone who is interested in a special phase of radiation biology or radiation therapy.

There are excellent illustrations and the print is of high quality.

For a practicing radiologist who can read German, the book is of considerable interest for a general review of basic biologic and therapeutic radiation problems. Some of the chapters may not be applicable for a busy practitioner of radiology, since they pertain to rather specific biologic radiation effects. The chapters on therapeutic radiology, however, are excellent reading for the radiation therapist who wishes to acquaint himself with the broad field of new means of irradiation as practiced in mid-European clinics and centers. The world literature pertaining to these separate fields (especially publications from the United States, all other European countries and Russia) is well assimilated and incorporated into the review of these special aspects of radiation biology and therapy. This book is highly recommended and should be available in any larger medical library as well as in the library of the radiobiologist or radiotherapist who can read German.

HANS W. HEFKE, M.D.

BOOKS RECEIVED

VERTEBRAL AND CAROTID ANGIOGRAMS IN TENTORIAL HERNIATIONS; INCLUDING ROENTGEN ANATOMY OF THE TENTORIAL INCISURE. By Hans F. Plaut, M.D., Chief, Radiology Service, Veterans Administration Center, Dayton, Ohio; Clinical Assistant Professor of Radiology, Ohio State University, College of Medicine, Columbus, Ohio; and Formerly, Instructor in Radiology, University of Cincinnati College of Medicine, Cincinnati, Ohio. Cloth. Price, $10.50. Pp. 155, with many illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

A MANUAL FOR NUCLEAR MEDICINE. By E. R. King, Captain, MC, U. S. Navy; and T. G. Mitchell, Lieutenant, MSC, U. S. Navy. Cloth. Price, $13.50. Pp. 406, with 80 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

PHLEBOGRAPHY AND VENOUS PRESSURE DETERMINATION; A STUDY INTO THE CORRELATION OF THE DATA GAINED FROM THESE METHODS. By M. N. Van Der Heyde, M.D. Cloth. Price, $5.00. Pp. 83, with 23 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

BRONCHOGRAPHIE CLINIQUE. By Étienne Bernard, Professeur de Clinique de Pneumo-Phtisiologie à la Faculté de Médecine de Paris, Médecin de l'Hôpital Laënnec; and Bernard Gamain, Chef de Laboratoire à la Faculté de Médecine de Paris, Assistant de l'Hôpital Laënnec. Paper. Pp. 196, with 132 illustrations. Masson et Cie, Éditeurs, 120

Boulevard Saint-Germain, Paris (6e), France, 1961.

Die juvenilen Rückgratverkrümmungen. By Priv.-Doz. Dr. Med. Friedrich Wilhelm Rathke, Heidelberg. Cloth. Price, $5.90. Pp. 156, with 122 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Hartstrahltechnik. By Dr. Med. Wolfgang Frik, Privatdozent der Med. Strahlenkunde, Med. Klinik mit Poliklinik der Universität Erlangen. Paper. Price, $7.15. Pp. 144, with 76 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Die Präoperative Röntgenbestrahlung des Mammakarzinoms. By Dr. E. Muntean, Universitätsdozent, Graz. Paper. Price, $4.40. Pp. 108, with 9 illustrations and 26 tables. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, New York.

Éléments de Radiobiologie Appliquée. By Roland Buchet, Médecin Radiologiste des Hôpitaux de Paris; avec la collaboration de G. Breitman, Attaché d'Électroradiologie des Hôpitaux de Paris. Paper. Pp. 189, with 127 illustrations. Masson et Cie, Éditeurs, 120 Boulevard Saint Germain, Paris (6e), France, 1960.

Acta Orthopaedica Scandinavica. Supplementum No. 43. Lumbar Intradiscal Pressure; Experimental Studies on Post-mortem Material. By Alf Nachemson. Paper. Pp. 104, with illustrations and tables. Ejnar Munksgaard, Copenhagen, Denmark, 1960.

Acta Orthopaedica Scandinavica. Supplementum No. 45. The Effects of Subperiosteallay Implanted Autogenous Whole-Thickness Skin Graft on Growing Bone; An Experimental Study. By Jaakko O. Elo. Paper. Pp. 90, with many illustrations and tables. Ejnar Munksgaard, Copenhagen, Denmark, 1960.

Acta Orthopaedica Scandinavica. Supplementum No. 46. The Surgical Anatomy and Pathology of the Supraspinous and Interspinous Ligaments of the Lumbar Spine with Special Reference to Ligament Ruptures. By Pentti M. Rissanen. Paper. Pp. 100, with 57 illustrations. Ejnar Munksgaard, Copenhagen, Denmark, 1960.

Acta Radiologica. Supplement No. 201. risa Encephalography and Conventional Neuroradiologic Methods; A Comparative Study. By Giovanni Di Chiro. Paper. Price, Sw.Kr. 35:–. Pp. 102, with many illustrations and tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1961.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. LEUCUTIA, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

HEAD

THIÉBAUT, F., WACKENHEIM, A., and VROUSOS, C. Nouvelles données de la pneumostratigraphie sagittale médiane de la fosse postérieure. (New data on median sagittal pneumostratigraphy of the posterior fossa.) *J. de radiol., d'électrol. et de méd. nucléaire*, Feb., 1961, *42*, 1–7. (From: Laboratoire de Radiologie de la Clinique neurologique de Strasbourg, France.)

In an earlier article published in the same journal in 1959, the authors described the laminagraphic technique applied to the study of the vermis cerebelli during a pneumoencephalographic examination. The normal and pathologic roentgen anatomy of this region is important in order to recognize some neurologic diseases and malformations, and to avoid unnecessary surgical exploration.

Three points are mentioned concerning the normal anatomy as seen roentgenographically: (1) dilatation of the postpyramidal groove is considered a normal variant in the absence of cerebellar symptoms and is not a sign of atrophy of the inferior vermis. It forms an accessory inferior cerebellar lobe containing the pyramis, the nodulus, the uvula and the tonsil; (2) the shadow of the vertebral artery is projected as an opaque curl in the foramen magnum and may be seen if the section is paramedian; and (3) diverticular dilatation of the vallecula may be seen in the posteroinferior part of the fourth ventricle.

In the pathologic roentgen anatomy, the authors first report some congenital malformations of the vermis. In agenesis of the inferior vermis, the superior part of the vermis (culmen) shows normal lobulation and the inferior part is hypoplastic and free of furrow formations, with an enlargement of the posterior cerebellar cistern. The authors have observed 5 of these cases which did not show cerebellar symptoms. In cases of Arnold-Chiari malformation with absence of the vermis, this absence may be predicted if the normal anterior transverse groove is not visible on the median convexity of the cerebellum and if, then, the furrows of the cerebellar convexity are seen as the spokes of a wheel, replacing the vermis. This absence of the vermis is also encountered in a rare malformation, cyclocerebellum, which is accompanied by other cerebral malformations.

Secondly, the authors discuss cerebellar atrophy. To distinguish hypoplasia of the vermis from an acquired atrophy, they believe that in the latter case the normal furrows are still present but are enlarged passively by parenchymal retraction. They report cases of cerebrocerebellar atrophy in which the atrophy of the vermis was either silent or clinically symptomatic. In 2 cases, they observed a combination of cortical parietal atrophy and atrophy of the vermis. In 1 of these 2 cases, the pneumoencephalographic signs of the cerebral atrophy were on the side opposite to the cerebellar atrophy.

In conclusion, the authors state that the laminagraphic studies permit recognition *in vivo* of some normal anatomic variants and malformations of the cerebellar vermis.—*H. P. Lévesque, M.D.*

SPITZER, R., RABINOWITCH, J. Y., and WYBAR, K. C. A study of the abnormalities of the skull, teeth and lenses in mongolism. *Canad. M.A.J.*, Mar. 18, 1961, *84*, 567–572. (Address: 3, Prince's Court, Brompton Road, London, S.W.3, England.)

A study was made of the dentition and lenses of 33 mongoloids and of the roentgen appearance of the skulls of 29 mongoloids who were patients at Leavesden Hospital, Herts, England. Their ages ranged from eight to forty-nine years.

There were no roentgen or clinical findings pertaining to the skull, teeth or lenses that were present in all cases. However, there was extreme thinness of the cranium (94 per cent); failure of development of the frontal sinuses (84 per cent); a high percentage of impaired development of *all* paranasal sinuses and mastoids; a high percentage of hypoplasia of the facial skeleton; dental changes, primarily microdontia and anomalies of shape (86 per cent); and a high percentage of lens opacities (23–85 per cent) of three main types, the percentage varying according to the type of opacification. There were other anomalies found less frequently.

The information gained from this study reveals that the anomalies do not occur sporadically, but form a pattern of defects. Since the defects involve a large range of tissues of different derivation, this suggests that the anomalies have been impressed on the developing fetus by genetic endowment rather than by prenatally acquired factors. The facts are suggestive of an inherent fault in the anlage of the structures affected, causing prenatal developmental interference.—*Gerald Davis, M.D.*

BLATT, NICOLAS, ZAMFIR, D., ATANASIU, M., and SLOBOZIANU, H. (Bucarest, Romania.) Les altérations du canal optique dans la maladie de Crouzon (dysostose craniofaciale). (Alterations of the optic canal in Crouzon's disease [craniofacial dysostosis].) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1960, *41*, 645–659.

In craniofacial dysostosis, compressive atrophy of the optic nerve may result from either intra- or pericanalicular hyperostosis; typically, the canal's vertical height is diminished by deformity of the lesser sphenoidal wing. In cases of concurrent oxycephaly, optic atrophy is especially likely to occur because of the added factor of intracranial hypertension.

Evaluation of the optic foramen has some prognostic value, since, if it is intact, the dystrophic process is benign, not only as it concerns the organs of vision but also the skull and brain.

Not all cases of craniofacial dysostosis show the imprint of cerebral convolutions on the calvarium. The authors note that in 1 of their cases a concurrent hydrocephalus prevented the development of these digital impressions.—*Frank A. Riebel, M.D.*

Legré, J., Denizet, D., and Savelli, J. (Marseille, France.) Lacunes craniennes secondaires à un traumatisme de la première enfance. (Cranial lacunae secondary to trauma suffered in early childhood.) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1960, *41*, 667–678.

Localized trauma in early childhood occurs in all cases of cranial lacunae; in 6 of the authors' 7 cases the injury occurred before the age of two. In 2 of the cases the parietal bones were affected as the result of the application of obstetrical forceps with resulting ischemic necrosis; the other 5 gave a history of falls.

The initial pathology largely determines the course; a dural tear is especially important, as it may permit development of a leptomeningeal cyst, porencephaly, or its healing may produce cicatricial retraction between bone and brain through the intermediary of a dense block of fibrous tissue. While usually a true fracture occurs, it is possible to have an intradiploic hemorrhage which causes progressive spreading of the two tables of bone. It is difficult to say when the lacuna develops because frequently there is a latent period between injury and the development of symptoms. In 1 of the cases cited, however, a leptomeningeal cyst apparently developed in a few days.

The typical roentgen findings are a clear lacuna in the cranial bone at the site of the old injury, with the outer table showing extensive exophytic reaction, often appearing like the crest of a helmet; the internal table may possess spicules. Usually the affected half of the skull is the larger, although in 1 of the authors' cases the opposite was true because of brain atrophy. A pneumoencephalogram will show slight dilatation of the ventricle on the affected side and its retraction toward the lacuna.—*Frank A. Riebel, M.D.*

Hurxthal, Lewis M. Pituitary gigantism in a child five years of age: effect of x-radiation, estrogen therapy and self-imposed starvation diet during an eleven-year period. *J. Clin. Endocrinol. & Metabol.*, Mar., 1961, *21*, 343–353. (From: Department of Internal Medicine, The Lahey Clinic, Boston, Mass.)

A case history of pituitary gigantism in a five year old girl is presented. The syndrome began at the age of three, and was studied for eleven years (age five to age sixteen). The syndrome is well documented, and reproductions of impressive skull roentgenograms and photographs of the child are included. Detailed metabolic data are given in tables.

At the age of nine, 2,400 r was administered to the pituitary by rotational supervoltage roentgen therapy. Further therapy was refused. The serum phosphorus and growth rate decreased for a period of about nine months. Further radiation therapy was not given, and estrogens were used in an attempt to control the child's growth. The data show that some further improvement was obtained; however, it was less striking than that following radiation treatment. At the age of twelve the patient placed herself on a starvation diet, and during this time she grew only one-fourth inch. No change in the sella was noted during the period of malnutrition (four years).

The author recognizes the fact that a larger dose of radiation should have been used if possible, and that it should have been employed when the child was first seen at the age of five. The fact that the roentgen therapy gave the best results again demonstrates its value in the treatment of pituitary eosinophilic adenomas. Serum phosphorus determinations are shown to be a useful adjunct in following these patients.—*William K. Littman, M.D.*

Wisoff, Hugh S., and Rothballer, Alan B. Cerebral arterial thrombosis in children; review of literature and addition of two cases in apparently healthy children. *A.M.A. Arch. Neurol.*, Mar., 1961, *4*, 258–267. (Address: 27 Ludlow St., Yonkers 5, N. Y.)

This is a report of 2 cases of cerebral arterial thrombosis in apparently healthy children, both diagnosed by angiography. The first patient died of extensive encephalomalacia and necropsy revealed areas of diffuse vascular disease involving both systemic and cerebral arteries, and a dissecting aneurysm almost totally occluding the lumen of the terminal internal carotid artery and the proximal middle and anterior cerebral arteries on the right. The second case survived with residual hemiparesis and dysphasia; there was considerable narrowing of the supraclinoid portion of the left internal carotid artery seen on angiography. The etiology in this case is obscure although there was a history of head trauma.

The authors also reviewed 27 cases of cerebral thrombosis in children under sixteen years of age, excluding those instances where the etiology and pathogenesis is already well recognized. The incidence seems to be rather low; however, they predict that with the increased use of angiography the disease will prove to be more common.

Males are more often affected and a definite left-sided preponderance is demonstrated. Clinically, an apoplectiform onset was more prevalent and head-

aches and seizures were fairly common. The site of the occlusion is correlated with the degree of recovery and the more proximal occlusions tend to be more benign. The cases of dissecting aneurysm of the internal carotid artery and middle cerebral arteries form the most significant group, although in the remainder of the cases no convincing etiology was found.—*Homero F. Nino, M.D.*

MEYER, H.-ST., BUSCH, G., and DÖRFFEL, E. W. Beitrag zum Problem des "Pseudotumor cerebri" und einer intrazerebralen Verkalkung. (Contribution to the problem of "pseudotumor cerebri" and an intracerebral calcification.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1961, *94*, 107–115. (From: Universitäts-Nervenklinik, Röntgenabteilung der Nervenklinik und Universitäts-Geschwulstklinik der Charité in Berlin, Germany.)

The authors take exception to the misused term "pseudotumor cerebri" when it describes any unsolved neurologic condition with increased intracranial pressure. One may find, for instance, the following lesions diagnosed as pseudotumor cerebri: brain abscess, subdural hematoma, aneurysm, atypical migraine, pregnancy encephalitis, thromboangiitis obliterans, tower skull, etc. Nonne coined this term in 1904 to describe subacute or chronic conditions with increased intracranial pressure, and papilledema without demonstrable brain tumor, even at autopsy. (The course is, as a rule, benign with complete remission.) Kehrer proposes to abolish this deceiving pseudodiagnosis and to use instead "increased intracranial pressure of unknown origin."Ketz considers the diagnosis of "pseudotumor cerebri" in certain cases as justifiable, although he agrees with Kehrer that it represents only a clinical syndrome. Some of his 17 observed cases show a normal ventriculogram; others demonstrate a distinct displacement appearance. Zuidema and Cohen, on the other hand, consider the syndrome "pseudotumor cerebri" to represent increased intracranial pressure with papilledema and a normal ventriculogram.

The authors propose to limit the term "pseudotumor cerebri" to the definition of Nonne except for the inclusion of a definite mass displacement visualized with cerebral arteriography and encephalography. To be valid, however, the latter finding must regress at the same time as the clinical symptoms without treatment.

The case report of a patient observed for ten years and fulfilling the above requirements is described. In their discussion the authors mention the very exceptional case of Binswanger, presented in 1912, in which, also, the tumor was not found at operation. Subsequently the patient's symptoms regressed considerably. However after twelve years an autopsy revealed a glioma the size of a hazelnut in the left temporal lobe. In the authors' case, the possibility of a circumscribed encephalitis, brain abscess, parasitic disease or tuberculoma was eliminated. An intracerebral hematoma, calcified after ten years, was believed to have been caused by the needle biopsy undertaken during the operation rather than to have been the cause of the original disease. The possibility of a thorium deposit secondary to thorotrast angiography producing the area of increased tissue density was ruled out with the help of a scintillation counter.—*S. Silvera, M.D.*

WORST, J. G. F. A röntgen reference contact lens, fixed by low vacuum. *J. belge de radiol.*, 1960, *43*, 591–597. (From: University Eye Clinic, Groningen, Holland.)

A contact lens which contains radiopaque markers imbedded in it is described. It is attached to the cornea by a low suction vacuum device. Anteroposterior and lateral roentgenograms are made on which a transparent reference scheme is superimposed. Intraocular foreign bodies are accurately located by this technique.—*David Morse, M.D.*

NECK AND CHEST

ISSA, PHILIPPE. Laryngocèle ventriculaire (à propos de 4 cas). (Ventricular laryngocele [report of 4 cases].) *J. de radiol., d'électrol. et de méd. nucléaire*, Feb., 1961, *42*, 8–16. (From: Hôpital de l'Université Américaine, Service de Radiologie, Beyrouth, Lebanon.)

This rare lesion was first described in 1829 by Larrey as a vesicular or aerial goiter. In 1867, Virchow coined the name "ventricular laryngocele." Thereafter, various names were used for its designation: aerial cyst, diverticulum or aerial hernia of the larynx, monkey pouch or gland.

Description. A laryngocele is an anomalous air sac arising from Morgagni's ventricle or, more precisely, from its anterior vertical extension in the arytenoepiglottic fold, which is 1 cm. long and is called the appendix, pouch, or sacculus ventriculi laryngis. This laryngeal pouch is the vestige of a large bilateral air sac, serving as an oxygen reservoir, which is found in anthropoid monkeys and other mammals and which extends through the thyrohyoid membrane to the base of the neck and sometimes to the axillary regions. In man, this saccule plays an important physiologic role in the lubrication of the vocal cords by its numerous mucous glands. The distention of this saccule results in a true hernia with a wall of its own and a neck attached to the anterior part of the ventricle. Its content is usually air; it may be liquid if drainage is hindered, or purulent with supervening infection, in which case it would be called a laryngopyocele.

Classification. Laryngoceles have been divided by

Lindsay into three categories: (1) internal, expanding in the laryngeal cavity above the superior border of the thyroid cartilage; (2) external, perforating the thyrohyoid membrane and bulging in the neck; and (3) mixed, which has both an external and an internal component. The last two types are the most frequent. Laryngoceles may be bilateral, generally of a different type on each side.

Pathogenesis. Two causes are considered. The first is a congenitally long sacculus with deficient thyroarytenoid muscle action, or a congenital weakness of the thyrohyoid membrane. The second cause is a consequence of increased intralaryngeal pressure which occurs during strenuous and repeated efforts such as singing, shouting, etc. Other contributing factors are local: tumor, granuloma or a cicatrix at the level of the ventricle with trapping of air in the saccule in a valve-like action.

Symptomatology. This varies from a long latency to an acute suffocating attack necessitating a tracheotomy. The most frequent symptoms are: (1) a slight change in voice, dysphonia or aphonia. This symptom is generally present with an internal laryngocele; (2) minimal dyspnea, in the case of slowly progressing enlargement of a laryngocele, or pronounced, even acute, dyspnea when distention is more rapid. It may be continuous, or provoked by effort; (3) the sensation of the presence of an accompanying foreign body with dry cough or the production of white mucoid or purulent secretions. At the acme of an attack, a vomica may occur; (4) rare and slight dysphagia; and (5) pain if the laryngocele is infected.

Clinical and Laryngoscopic Findings. An evocative sign of external laryngocele is a mass located in the anterior triangle of the neck above the thyroid cartilage. It may give an impression of fullness without clear delineation. This is also produced by an internal laryngocele repelling the thyrohyoid membrane. The mass may be big and may extend to the supraclavicular region. It is rounded or lobulated, regular, soft, and tympanic on percussion. It may change in size with coughing and may empty on pressure. When the neck of the sac is obstructed, the mass becomes very hard.

On endoscopic examination of an internal laryngocele, a pinkish rounded mass is seen which is depressible and occupies the lateral portion of the laryngeal vestibule, extending to the aryepiglottic fold and sometimes to the epiglottis or the base of the tongue.

Roentgen Findings. Roentgen examination is important in establishing the clinically suspected diagnosis or in confirming a clinical impression by showing the typical aspect of a laryngocele. On the anteroposterior roentgenogram, a well-defined air shadow will be seen in the para- or intralaryngeal location. The first air collection will be found in the soft tissue of the neck, the second air pocket will be seen through the cervical spine, not extending below the level of C5–C6. On the lateral roentgenogram, the shadow of a laryngocele will be superimposed on the larynx and will extend above it, but its lower margin will not go beyond the level of Morgagni's ventricle. The distance between the hyoid and thyroid cartilages may be increased by compression from the mass, or these structures may show local displacement by it.

Frontal plane laminagraphy will clearly show the two gaseous pouches with a narrow channel connecting them. A fluid level will be seen if the laryngocele is partly filled with secretions.

The author reports 4 cases in full detail with illustrative roentgenograms. In the differential diagnosis, he discusses mucous laryngeal cysts, posttraumatic hemorrhagic cysts of the vocal cords, and congenital cysts from branchial inclusions or cells of the laryngeal saccule. The only curative treatment is the surgical removal of the mass with its wall.—*H. P. Lévesque, M.D.*

Dalith, F. Calcification of the aortic knob: its relationship to the fifth and sixth embryonic aortic arches; a radiological and anatomical study. *Radiology*, Feb., 1961, *76*, 213–222. (Address: Department of Radiology, Government Hospital, Tel-Hashomer, Israel.)

In order to identify the site of preference of calcium deposition in the aortic arch, to investigate the nature of this finding, and to explain its roentgen appearance, laminagraphic examinations in the anteroposterior and latero-oblique projections were made in 27 patients in whom a calcium-dense opacity at the aortic knob was demonstrated on a routine chest roentgenogram. In addition, 100 aortic arch specimens were obtained from consecutive autopsies, fixed in formalin, studied roentgenographically, subjected to gross macroscopic examination, transilluminated and photographed, and studied histologically.

Laminagraphic studies demonstrated that the calcium-dense opacities were practically always situated in the wall of the aorta in the mid portion of the aortic arch opposite the shadow of the pulmonary artery, suggesting that they might be related to and appear as a sequel to the closure of the ductus arteriosus. In the studies of the autopsy specimens of the aortic arch, calcification of the aortic knob was found to be the result of single plaques in the aortic wall located either at the entrance of the ligamentum arteriosum or at the level of the origin of the left subclavian artery, or at both sites. Each of these areas showed a depression on the intimal surface, with increased translucency when viewed by transillumination, indicating thinning of the aortic wall. It may be concluded, therefore, that the preferential deposition of calcium in the aortic arch is related to these structural changes which correspond to the

aortic connections of the sixth and, most probably, the fifth embryonic aortic arches.

The significance of the roentgenologic manifestation "calcified aortic knob," *per se*, for generalized atherosclerosis of the aorta is questioned. Its presence in young persons with patent ductus arteriosus may be accepted as a confirmatory sign of that anomaly. —*Walter H. Jarvis, Jr., M.D.*

SCHEMINZKY, F., and SCHRÖCKSNADEL, H. Einfluss der Radiumemanation auf Herzfrequenz und Blutdruck. (Influence of radium emanation upon pulse and blood pressure.) *Wien. klin. Wchnschr.*, Feb. 17, 1961, *73*, 123–127. (From: Forschungsinstitut der Oesterreichischen Akademie der Wissenschaften in Badgastein und Physiologisch-Balneologisches Institut der Universität, Innsbruck, Austria.)

Observations on the effects of combined hyperthermy and radon inhalation upon pulse and blood pressure in more than 10,000 patients have been made by many competent investigators in the deep thermal galleries of Badgastein. Temperatures were around 42° C.; the relative humidity was 100 per cent and the concentration of the radon about 4.1×10^{-9} curie per liter. The combined effect of humid air at a high temperature and radon inhalation produced in a large number of patients a considerable reduction of strain in blood circulation.

Since such studies in the Badgastein galleries are subject to many variable and often uncontrollable factors, the authors made similar studies under better controllable conditions in the artificial emanatorium of the Innsbruck Physiologic-Balneologic Institute. The pulse and blood pressure of 45 healthy medical students were measured before and after the inhalation of radon (at a concentration of 5×10^{-9} curie per liter) for one hour. Inhalation of this gas at room temperature had practically no effect upon pulse and blood pressure. However, if the room temperature was increased to 41.5° C. and the humidity raised to 90 per cent, it was observed that radon inhalation considerably reduced the strain upon blood circulation produced by the hyperthermic conditions. No explanation of the mechanism of this specific radon effect can be offered at present.—*Otto Glasser, Ph.D.*

VON BRAMANN, CONSTANTIN. The surgical treatment of primary and secondary reflux esophagitis. *Proc. Rudolf Virchow Med. Soc.*, 1960, Jubilee Volume, *99*.

Primary reflux esophagitis is most frequently found in cases of diaphragmatic hiatus hernia. Secondary reflux esophagitis occurs after surgery on the cardiac portion of the stomach, particularly after resection of the cardia for ulcer or carcinoma. The most painful form of reflux esophagitis is observed after anastomosis between the esophagus and the jejunum (esophagojejunostomy).

The numerous operative procedures recommended for treatment of primary and for prevention of secondary reflux esophagitis are described. However, it is also pointed out that the majority of esophagogastric anastomoses do not lead to reflux esophagitis, even without preventive operative techniques. In the author's own series of about 100 cardiac resections, there were only 2 patients who developed reflux esophagitis and these responded to conservative measures.

Even with this low incidence, reflux esophagitis is a real problem to the surgeon, internist and general practitioner because of its severe and disabling symptoms and because of the steadily increasing number of operations on the lower esophagus and cardiac portion of the stomach. The chances for the future development of ways for successfully replacing the esophagus with plastic material are considered to be favorable.—*Franz J. Lust, M.D.*

ABDOMEN

HEALEY, WILLIAM V., RIGGINS, ROBERT C., WHITE, RICHARD L., HABIF, DAVID V., and STEWART, WELLINGTON B. An experimental method for localizing the source of intestinal bleeding. *Surg., Gynec. & Obst.*, Mar., 1961, *112*, 285–288. (From: The Departments of Surgery and Pathology, College of Physicians and Surgeons, Columbia University, New York and Department of Pathology, College of Medicine, University of Kentucky, Lexington, Ky.)

An experimental method for localization of gastrointestinal hemorrhage is described. In this technique, canine red blood cells were labeled with radioactive P^{32}. A series of 3 small Geiger-Müller counters was placed at 8 inch intervals within a Cantor tube passed into the intestine. It was possible to detect local beta radiation with the counter nearest to the bleeding site. There were no false positive readings in the 7 dogs tested. No more than 15 cc. of blood was used in any test segment. Multiple bleeding sites were effectively localized by this method.

The concept is a sound one and, with further technical improvements, the method may find useful clinical application.—*Bernard Loitman, M.D.*

DALLOS, ARTHUR. Complete intestinal obstruction by Meckel's diverticulum. *Proc. Rudolf Virchow Med. Soc.*, 1959, *18*, 76.

A forty-nine year old man with frequent vomiting and severe abdominal pains was admitted to the hospital for a possible intestinal obstruction. A flat roentgenogram of the abdomen confirmed the

mechanical obstruction. After a short period of deflation and decompression, the patient was operated on. The lower ileum was completely obstructed by a kinking which was caused by a sizable Meckel's diverticulum (approximately 2½ inches long). A small bowel resection including the kinking diverticulum was done. The proximal ileum was anastomosed to the cecum.

Meckel's diverticulum represents a part of the omphalomesenteric duct. A free, blind diverticulum to the ileum is the most common form of the anomaly. It usually occurs within one meter of the terminal ileum.

A Meckel's diverticulum is prone to be involved in three types of pathology: (1) infection, (2) intestinal obstruction, and (3) peptic ulcer. Acute diverticulitis is similar to the clinical picture of acute appendicitis. The obstruction can take the form of kinking, torsion, or constriction of the small intestines or, more rarely, of a mobile portion of the colon. Intussusception may occur. A peptic ulcer in a Meckel's diverticulum may be complicated by perforation, obstruction, or hemorrhage. The presence of a Meckel's diverticulum can seldom be diagnosed before operation.—*Franz J. Lust, M.D.*

Elkan, Wolf. Two rare tumors of the ileum. *Proc. Rudolf Virchow Med. Soc.*, 1959, *18*, 81.

Tumors of the ileum are extremely rare, their frequency among abdominal tumors being estimated as less than 1 per cent. They are difficult to diagnose, since the liquid nature of the contents of the small intestine makes obstruction a very late manifestation and the mobility of the small intestine allows them to move about more freely and thus prevents early manifestation of pain due to pressure on neighboring structures. Early roentgen diagnosis is equally difficult because of the irregular pattern and the superimposition of the small intestine.

The first patient reported was a seventy year old female. She had first noticed progressive weakness, loss of weight and appetite, and abdominal distention. Later, nausea and vomiting occurred and she consulted her physician, who discovered a large, fixed mass in the right pelvic region. At operation, a large adenocarcinoma of the ileum was found.

The second case was a sixty year old male who suffered from hypertension (about 200), chest pains, headaches, profuse perspiration, flushing of the face and a feeling of weakness. At first the only positive findings were a sedimentation rate up to 130; however, on roentgen examination of the gastrointestinal tract, an oval filling defect was noted in the right lower abdomen indicating a mass displacing the intestine. At operation, a tumor of the ileum was found; it was a carcinoid of the ileum.—*Franz J. Lust, M.D.*

Margulis, Alexander R., and Mandelstam, Paul. The use of parenteral neostigmine in the roentgen study of the small bowel. *Radiology*, Feb., 1961, *76*, 223–229. (Address: 510 South Kingshighway, St. Louis 10, Mo.)

This paper describes a method of obtaining rapid and good visualization of the small bowel with the use of neostigmine methylsulfate (prostigmin, Roche), which acts as a reversible inhibitor of acetylcholinesterase. This action increases small bowel motility and, therefore, the speed of intestinal transit. Over 3,000 examinations of the small bowel with neostigmine have been completed during a period of ten months at the Mallinckrodt Institute of Radiology. There were virtually no untoward results and in no case was medication or cessation of the examination necessary. No myocardial infarctions, intestinal perforations, or other serious complications occurred that could be attributed to the use of this drug. The contraindications in the dose range used by the authors are: intussusception, volvulus, complete intestinal osbtruction, perforation, peritonitis, and recent myocardial infarction.

An initial pilot study in 107 successive unselected patients who received 0.5 mg. (or 0.75 mg. in patients weighing more than 190 lb.) neostigmine subcutaneously after completion of fluoroscopy of the stomach and duodenum showed that barium had reached the cecum or beyond within one hour in 93 (87 per cent). A subsequent study was done in which 153 patients were given neostigmine as above; another 299 patients were given a placebo (an equal volume of 5 per cent glucose in water) subcutaneously, and another 157 patients received no injection. Neostigmine was shown beyond doubt to speed intestinal transit. In comparison with the control group receiving no injection, the placebo definitely accelerated propulsion of the barium meal, although it was not nearly as effective as neostigmine, and also showed the importance of psychic influences on intestinal motility.

The procedure now followed in routine upper gastrointestinal surveys (in the absence of contraindications) is to give 0.5 mg. of neostigmine (0.75 mg. in patients over 190 lb.) subcutaneously following fluoroscopic examination of the stomach and duodenum, with roentgenograms being taken fifteen and sixty minutes after the injection and, if necessary, later. When detailed studies of the small bowel are desired, roentgenograms are obtained at fifteen, thirty, forty-five, sixty, and one hundred and twenty minutes after administration of neostigmine and at further intervals if necessary.

The authors have been able to diagnose by these means cases of sprue, lymphosarcoma of the small bowel, regional ileitis, adhesions, and involvement of the small bowel by metastatic tumor. The drug does not modify the characteristic morphology and does not alter transit in conditions of disordered motor function.—*Donald N. Dysart, M.D.*

GRIFFITHS, J. D. Extramural and intramural blood-supply of colon. *Brit. M. J.*, Feb. 4, 1961, *1*, 323–326. (From: St. Bartholomew's Hospital, London, England.)

Postmortem injection studies of blood vessels can be misleading due to technical difficulties such as postmortem clotting, spasm and the viscosity of the injection solution. For the studies of the colon reported by the author, the specimens were injected as soon as possible after death or surgical removal. Infusion with a weak ammonia solution or a saline heparin solution prior to injection prevented necropsy spasm and postmortem clotting, respectively. A barium gelatin or a barium methylcellulose mixture was used as the medium for visualization. A series of experiments was performed which demonstrated the anastomoses between the superior and inferior mesenteric arteries via the middle and left colic arteries. They showed that ligation of the inferior mesenteric artery is a feasible procedure. The adequacy of the marginal artery as a collateral channel and the presence of adequate anastomoses between the middle rectal and superior rectal arteries were shown. Submucosal, mucosal and muscular plexuses form an intricate pattern in the wall of the colon. It is important that ligation of the marginal artery at surgery be at or about 2 cm. beyond the site of resection of the colon or ischemia will result.—*David Morse, M.D.*

KHILNANI, MANSHO T. Calcified liver metastasis from carcinoma of the colon. *Am. J. Digest. Dis.*, Mar., 1961, *6*, 229–232. (From: Department of Radiology, Mount Sinai Hospital, New York, N.Y.)

The appearance of the fine, punctate, closely spaced calcifications in necrotic areas of liver metastases from carcinomas of the colon has been well described previously in cases with the metastases usually coming from the sigmoid or rectosigmoid portion of the colon. The present report is mainly concerned with similar metastases from a carcinoma of the right colon, but it does add another case in which the metastases came, characteristically, from the sigmoid.

This type of calcium deposition somewhat resembles the so-called psammoma calcification characteristic of ovarian carcinoma, but it can readily be distinguished from the peripheral ring-like calcification associated with cysts or abscesses of the liver, the multiple discrete calcifications of disseminated tuberculosis or histoplasmosis, and the phleboliths of a hemangioma. The mottled appearance of thorotrast deposition is more diffusely spread through the entire liver and includes the spleen. A primary hepatoma can show calcifications similar to those in a metastasis from the colon but, as these are usually found in young children, the discrepancy in the ages of the two groups makes the differentiation possible.

The calcified metastases from carcinoma of the colon do not indicate any special characteristics of the original tumor in contrast to the instances of calcification within primary gastric tumors that appear to be associated with mucoid types of carcinoma.—*Donald M. Monson, M.D.*

SICHEL, D., WOLFF, R., and EISENBETH, R. (Strasbourg, France.) Dépistage et fréquence des tumeurs benignes de la vésicule biliaire (étude de 20 cas). (Detection and incidence of benign tumors of the gallbladder [study of 20 cases].) *J. de radiol., d'électrol. et de méd. nucléaire*, Feb., 1961, *42*, 17–24.

Among the multiple causes of biliary dyskinesia and noncalculous cholecystopathy, one is particularly disregraded, even ignored, and that is the existence of benign tumors of the gallbladder.

Contrary to present day belief, these tumors are not rare. In less than two years, the authors have diagnosed 20 cases of benign tumors. Kirklin reported an incidence of 8.5 per cent in 17,000 surgical specimens. Jutras, Longtin, and Lévesque have recorded an incidence of 6.8 per cent of cholesterolosis in 2,494 removed gallbladders during a five year period (1951 to 1955).

Many reasons explain why these lesions escaped roentgenologic detection. A small tumor defect may go unrecognized due to the too great opacity of the gallbladder or it may be interpreted as an artifact or, especially, as intestinal gas. A precise roentgen technique is of prime importance. It should include roentgenograms taken before and after a fat meal and in different positions (erect, Trendelenburg, horizontal) with various angles of projection (en face, lateral, oblique, tangential). Gradual compression under fluoroscopic control is most important.

The main criterion in establishing the existence of a tumor is its fixation to the gallbladder wall, thus eliminating the possibility of gas or a stone.

The size of the tumor may vary from a few millimeters to a centimeter. The solitary defect has a rounded and regular contour when it is benign. The authors have not observed concomitant lithiasis. Very often the contour of the gallbladder is dentate, an appearance which corresponds to that of diffuse granular cholesterolosis.

The benign tumors of the gallbladder are usually a product of a hyperplastic cholecystosis. Jutras, Longtin and Lévesque have classified the hyperplastic processes into three main groups: (1) cholesterolosis (diffuse or strawberry gallbladder, solitary or multiple polyps of cholesterolosis), (2) adenomyomatosis, and (3) neuromatosis. If the tumor is not found on the specimen by the surgeon or the pathologist, it is because of faulty technique in the removal of the gallbladder, especially by clamping the fundus.

The authors do not state the total number of removed gallbladders, nor do they say if their 20

cases were confirmed. The roentgen appearance and histologic nature of the tumors are not described. Jutras and his associates have shown the high incidence of lithiasis with these hyperplastic cholecystoses.—*H. P. Lévesque, M.D.*

Lauchenaur, Clara. Zur Frage der Prophylaxe von Biligrafin-Zwischenfällen. (Prophylaxis of biligrafin reactions.) *Schweiz. med. Wchnschr.*, Jan., 1961, *91*, 45–46. (From: Universitätsinstitut für Röntgendiagnostik und Strahlentherapie des Bürgerspitals Basel, und der Pathologisch-anatomischen Anstalt der Universität, Basel, Switzerland.)

Intravenous administration of biligrafin (cholografin) for cholecystography frequently provokes hot flushes, sneezing, cough, urticaria, slight nausea and vomiting. These side effects are believed to be caused by a hypersensitivity to the entire molecule complex of the contrast material when an iodine allergy has been excluded. Severe reactions have become rare, but a case with fatal outcome is reported.

An ambulatory male patient, aged sixty-one, with the diagnosis of gallbladder disease, was referred for intravenous cholecystography to be followed by a gastrointestinal examination. He was cachectic and appeared very sick. After all preliminary tests for iodine sensitivity were made and proved negative, 20 cc. of biligrafin forte was injected intravenously within three minutes. Nausea ensued after the injection of 16 cc., but following a few deep respirations the injection could be completed. Thirty seconds later, however, the patient became very pale and began to have convulsions. This was followed by Kussmaul's respiration and cardiac arrest in systole. Thoracotomy was performed without delay, but cardiac massage proved ineffective.

Autopsy findings included aortic stenosis, coronary sclerosis, multiple myocardial scars, dilatation and hypertrophy of the left and right heart chambers, a patent foramen ovale, arteriosclerosis and atheromatosis of the aorta and its branches, a large adenocarcinoma of the gastric cardia with esophageal stenosis and metastatic spread, hydrops of the gallbladder secondary to an occluding cystic duct calculus and acute as well as chronic generalized congestion, hyperemia of meninges, cerebral edema, and evidence of moderate cerebellar tamponade.

Aortic stenosis may lead to sudden death. In this case, however, the sudden demise was undoubtedly due to the biligrafin injection. Since iodine sensitivity could be excluded, the reaction was believed to be due to calcium imbalance. The aortic stenosis may have caused prolonged stagnation of biligrafin in the dilated left ventricle. Thus, the cardiac musculature could have been suddenly deprived of vital calcium molecules. Recent animal experiments tend to corroborate such a conclusion.

In a series of 180 cases examined after the fatality described above, injection of calcium salts together with an antihistaminic drug was given prophylactically, and the subsequent intravenous administration of biligrafin was done very slowly. This premedication succeeded in preventing side effects. It must be remembered, however, that intravenous medication with calcium salts is contraindicated in patients being treated with digitalis.—*Ernest Kraft, M.D.*

Lamprecht, Werner. Pancreatic cysts in modern surgery. *Proc. Rudolf Virchow Med. Soc.*, 1960, Jubilee Volume, *274*.

The etiology, pathogenesis, symptomatology and diagnosis of pancreatic cysts are discussed. Only pseudocysts—those not lined with secreting epithelium—cause symptoms (usually because of their size) and require treatment.

Only surgical intervention is effective. Various operative procedures are evaluated and a new, improved operation is described which is simple, safe and satisfactory. It consists of a wide anastomosis between the cyst and the posterior wall of the stomach by a transcystic approach. The contents of the cyst—fluid and pancreatic tissue debris—are completely removed and the already existing adhesions between stomach and cyst are utilized in securing the anastomosis. The operation has been done on 8 patients and has resulted in roentgenologically proved fast shrinkage and obliteration of the cyst.

Clinically, it is important to watch for diabetes; its incidence in these 8 patients rose from 12.5 per cent preoperatively to 55.6 per cent postoperatively. —*Franz J. Lust, M.D.*

Gynecology and Obstetrics

Singleton, Edward B., Rosenberg, Harvey M., and Samper, Luis. Radiologic considerations of the perinatal distress syndrome. *Radiology*, Feb., 1961, *76*, 200–212. (Address: Texas Children's Hospital, Houston, Texas.)

The purpose of the present paper is to discuss the roentgen manifestations of the conditions which, in the opinion of the authors, are the sequelae of hypoxia or anoxia in the perinatal period, *i.e.*, directly prior to delivery, during the birth process, or immediately thereafter. These conditions, having a common denominator of anoxia and a common pathogenesis, *i.e.*, increased capillary fragility, constitute the perinatal distress syndrome. A number of them are roentgenologically demonstrable, whereas others are disclosed only at the time of necropsy.

Intracranial hemorrhages are commonly encountered in infants who have suffered from anoxia in intrauterine life or in the neonatal period. Although the degree is seldom severe enough to produce roentgen changes in the skull, intracranial hemorrhage should be suspected in any newborn infant

with respiratory distress and a normal chest roentgenogram.

Infants with hyaline membrane disease are usually born prematurely under circumstances consistent with anoxia, which by increasing capillary permeability may readily allow the effusion of fibrinogen into the alveoli and provide the necessary conditions for the formation of hyaline membranes. The roentgen features are usually characteristic and frequently present before the respiratory difficulties become noticeable.

Whereas hyaline membrane disease is more common in the premature infant, the aspiration syndrome is usually found in the postmature baby, apparently the result of the onset of respiration *in utero* or during passage through the birth canal. The respiratory symptoms are similar to those of hyaline membrane disease; however, the roentgen findings are similar to those in other forms of aspiration pneumonia. Pneumothorax and pneumomediastinum are common complications in the severe cases.

Pulmonary hemorrhage in the newborn may occur in small, clinically insignificant amounts or may at times be massive, resulting in rapid demise. Fetal anoxia is the most probable cause, although this may be complicated by hypothrombinemia or hemorrhagic disease of the newborn, or accompany erythroblastosis fetalis. Roentgenograms of the chest show changes similar to those in infants with the aspiration syndrome.

Localized noninfectious enterocolitis of early infancy may represent damage to the bowel produced by a focal area of vascular insufficiency. The condition may make its appearance at birth in the form of a localized ulceration of the esophagus, stomach, small intestine, or colon. With perforation, complications become obvious. In the absence of perforation, healing gradually occurs and the accompanying cicatricial changes may produce obstruction several weeks later.

Although intestinal atresia is generally accepted as being the consequence of localized incomplete recanalization of the gut between the seventh and eleventh weeks of embryonic life, there is evidence suggesting that some forms result from interruption of the fetal blood supply to the atretic area due to certain anoxic factors. The roentgen features consist of gaseous distention of the intestine promixal to the site of obstruction and multiple air-fluid levels, usually on upright or decubitus roentgenograms.

The livers of infants who have suffered from anoxia may show changes which are apparently unique to perinatal distress. These anoxic changes are not demonstrable by roentgenographic methods but serve to illustrate the danger of anoxia to the fetal organs. Hemorrhages into the adrenal gland may also result from anoxia and the consequent increase in capillary permeability. Calcification in the adrenal glands of older otherwise normal children probably represents the reparative end result of this condition. —*Walter H. Jarvis, Jr., M.D.*

GENITOURINARY SYSTEM

RUTNER, ALVIN B., and SMITH, DONALD R. Renal papillary necrosis. *J. Urol.*, Apr., 1961, *85*, 462–470. (From: The Division of Urology, University of California School of Medicine, San Francisco, Calif.)

Renal papillary necrosis is an uncommon lesion involving one or both kidneys. It is almost always associated with urinary tract infection and the patient may have a concomitant diabetes mellitus, obstructive uropathy, or chronic interstitial nephritis. In some instances, the disease has been associated with overdosage of phenacetin. The clinical course may be acute, subacute, or chronic. The primary pathologic lesion is a coagulative necrosis of one or more renal papillae with more or less involvement of the medulla. The cortex is not involved in the necrotic process but may show pathologic features of chronic nephritis secondary to the papillary disease. When associated with phenacetin abuse, the kidneys show diffuse interstitial fibrosis with a lymphocytic cellular infiltrate and with irregular contraction of the renal parenchyma.

A roentgen diagnosis can be established in the majority of subacute and chronic cases. The roentgen finding consists of a diffuse, hazy outline of the minor calyces in the early stages. Subsequently, as the disease progresses, there is evidence of sinus tract formation and the contrast medium may be seen in arc shadows or ring shadows adjacent to the minor calyces. These changes are observed both on the retrograde and intravenous pyelograms. They tend to be progressive and, in the final stages, show evidence of clubbing of the calyces with medullary necrosis and small contracted kidneys. During the course of the illness, the patient frequently passes necrotic tissue in the urine.

The prognosis is generally poor. Vigorous conservative management is indicated in most instances.

Ten figures consisting of anatomic drawings, reproductions of intravenous and retrograde pyelograms, and pathologic sections illustrate 4 case histories presented in this article.—*George W. Chamberlin, M.D.*

KLEIN, RICHARD L., and MCCHESNEY, JOHN A. Hypertension secondary to aneurysm of the renal artery: report of a case of long duration cured by nephrectomy. *Ann. Int. Med.*, Feb., 1961, *54*, 292–300. (From: Department of Medicine, Washington University School of Medicine, Barnes Hospital, St. Louis, Mo.)

The case of a twenty-six year old female is presented to illustrate an apparent cure of long-standing hypertension associated with an aneurysm of the left renal artery. The patient had been in excellent health until 1944, when, at the age of thirteen, she suddenly developed headaches accompanied occasionally by nausea and vomiting. The persistence of

these symptoms necessitated hospitalization, at which time her systolic blood pressure was 180 mm. Hg. She was treated with medication and received symptomatic relief. In 1951 she was admitted to another hospital for evaluation of hypertension. All laboratory tests, performed at this time, were within normal limits.

Urinary function tests, medications and the results are given for hospital observations in 1952 and 1956. These were during her two pregnancies, the first being a spontaneous delivery of a normal child at seven and one-half months' gestation and the second pregnancy terminating with the delivery of a seven month stillborn.

In 1957, differential renal function studies were performed, utilizing bilateral ureteral catheters. These showed a reduced glomerular filtration rate per nephron in the left kidney, compatible with extrinsic renal arterial disease. These observations were significant in that the combined glomerular filtration rate of both kidneys was within normal limits. An excretory urogram showed decreased concentration of the opaque medium by the right kidney and normal concentration on the left. Both kidneys were of normal size and shape.

An aneurysmal dilatation of the inferior branch of the left renal artery was demonstrated by translumbar aortography.

The patient has been normotensive for eighteen months following removal of the left kidney.— *John D. Hummel, M.D.*

WINTER, CHESTER C. Advances in the radioisotope renogram test. *J. Urol.*, Apr., 1961, *85*, 683–687. (From: The Department of Surgery, Division of Urology, Ohio State University Health Center, Columbus, Ohio.)

Since its introduction in 1956, radioisotope renography has proved its value as a screening and diagnostic procedure in the evaluation of a variety of urinary tract diseases. The renogram has received widest clinical acceptance in the evaluation of differences in renal vascular supply in the hypertensive patient in whom a nonrenal hypertensive etiology has been eliminated. The study is of value in detecting and following obstruction in the upper urinary tract; an ideal way of following a small ureteral calculus is with periodic plain roentgenograms and renograms. These show the position of the stone as well as the function and drainage of the involved kidney without the added exposure and risk of repeated excretory urograms. By renography one can also readily differentiate acute renal failure from complete obstructive uropathy in the anuric patient within the first forty-eight hours.

The author emphasizes the importance of attention to details and proper instrumentation in performance of the study. He feels that most nondiagnostic radioisotope renograms result from improper technique rather than from an inherent deficiency in the procedure itself. Hippuran I^{131} presently seems to be the test agent of choice because it is removed at a rapid rate from the blood exclusively by the kidneys.

By the addition of a third set of instruments to the dual renogram unit for monitoring the chest or any other highly vascular area, an estimation of the rate of blood clearance of hippuran I^{131} may be otbained. The author believes that this blood clearance curve is helpful in estimating quantitative total renal function.

Four short case presentations illustrate the clinical usefulness of this study.—*George W. Chamberlin, M.D.*

MACLAURIN, JOHN C., and ARNEIL, GAVIN C. Phaeochromocytoma causing ureteric obstruction in a child. *Lancet*, Feb. 25, 1961, *1*, 426–429. (From: The Department of Child Health, University of Glasgow, and The Royal Hospital for Sick Children, Glasgow, Scotland.)

The view is now taken that any adrenalin- or nonadrenalin-secreting tumor, irrespective of its site of origin, should be called a pheochromocytoma. Only 5 per cent of all reported cases of this tumor have been in children and, in them, 15 per cent have originated outside the adrenal gland.

This case report is of a twelve year old girl with an eight month history of intermittent headaches, one month of anorexia and some weight loss, and three recent episodes of left abdominal pain associated with nausea and vomiting, but no urinary symptoms, paroxysmal flushing, sweating or palpitation.

Investigative studies revealed that the facies and general demeanor vaguely suggested thyrotoxicosis, though exophthalmos and tremor were not present. Blood pressure on admission was 185/125 mm. Hg. There was bilateral papilledema, proteinuria, a four- to sixfold increase in the catecholamine content of the urine, an erythrocyte sedimentation rate of 59 mm. in the first hour and a solitary gallstone. Intravenous pyelography disclosed a nonfunctioning left kidney and at cystoscopy the ureteral catheter could not be passed more than 2 cm. up the left ureter. The paroxysmal nature of the hypertension became apparent with observation, but the Regitine test was equivocal. Notwithstanding, the correct diagnosis was suggested preoperatively.

At surgery, a tumor mass was found encircling the left ureter about 2 cm. above the bladder, but it was mainly around the lower inch of the aorta, the bifurcation and the left common iliac artery. The bladder wall was not involved. All tumor tissue was removed macroscopically with considerable difficulty. The left kidney and adrenal gland were also excised, the former because the degree of hydronephrosis was greater than expected, and the latter because clamping of the left renal vein caused an immediate drop in blood pressure. By palpation the right adrenal was

normal, and the left subsequently proved to be normal also. The postoperative course was satisfactory and her full recovery dramatic.

The problem of malignancy arises often since many of these tumors, as in this case, appear locally invasive. The authors concur that distant metastasis appears to be the only reliable criterion of malignancy, with such a complication present in about 2.5 per cent of cases.

It is speculated that the site of origin of this tumor was probably the organ of Zuckerkandl. Abdominal symptoms have been noted as being the rule in these cases. In the only other known case presenting with a nonfunctioning kidney, the tumor arose in the bladder.—*Donald M. Monson, M.D.*

NERVOUS SYSTEM

SCHOBER, R. Zystische Bildungen der häutigen Rückenmarkshüllen. (The formation of cysts in the coverings of the spinal cord.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1961, *94*, 116–129. (From: Universitätsstrahleninstitut, Homburg-Saar, Germany.)

The development of cysts in the space between the spinal cord and the spine produces symptoms of a space occupying lesion similar to disk herniation or to a tumor. These cysts are differentiated as intradural, extradural or epidural-perineural according to their location.

The author's series comprises 247 cases studied by myelography of the cervical region (49 cases), the thoracic region (36 cases), and the lumbosacral portion of the spine (162 cases). Twenty-one cases showed signs of epidural-perineural cysts, 16 in the lumbosacral region and 5 in the cervical region. These cysts are connected with the subarachnoid space and fill with the contrast medium as do diverticula. Their size varied from that of a peppercorn to a peanut. In none of the 21 cases could the neurologic symptoms be correlated with the myelographic findings. Eleven of the 16 cases of cysts in the lumbosacral region showed, in addition, evidence of herniation of the nucleus pulposus and in all these cases the cysts were below the level of the herniation.

In analyzing these findings, and reviewing the anatomy and embryology of the spinal cord, nerve roots and dural coverings, the author arrives at the conclusion that developmental abnormalities are a factor responsible for these cysts, a theory which seems especially plausible when one considers the shifting of the ganglia from the intradural to the extradural space and back again as observed in the 24 mm., 34 mm. and 54 mm. fetus. Also, one must take into consideration the disproportionate development in the length of the spine in relation to its contents from the time the fetus reaches 34 mm. For instance, root pouch of unusual length and varied width may develop if the coordination of length development to the movement of the ganglia is impaired.

A second factor in the development of epidural-perineural cysts is the pressure fluctuation of the cerebrospinal fluid, which is elevated when the pressure in the veins is raised thus causing decreased fluid absorption. This is especially true in herniation of the nucleus pulposus or other space occupying lesions in the extradural space which causes a localized circulation disturbance.

As a rule, these not infrequently observed epidural-perineural root pouch cysts have no clinical significance.—*S. Silvera, M.D.*

SKELETAL SYSTEM

INNES, JAMES, and NEWALL, JOSEPH. Myelomatosis. *Lancet*, Feb. 4, 1961, *1*, 239–245. (From: Departments of Medicine and Radiotherapy, University of Edinburgh and Royal Infirmary of Edinburgh, Scotland.)

One hundred eighteen cases of myelomatosis were reviewed in a study of the natural history of the disease. In this series the ratio of men to women was about 3 to 2 and the disease developed after the age of fifty in 80 per cent of the cases. The disease results from an abnormal proliferation of reticulum cells in or outside the bone marrow and is manifested by four different varieties.

Solitary Myeloma (46 cases). The plasma cell proliferation may start in a single bone, giving rise to a solitary myeloma. This eventually shows as a local swelling of bone which is often painless. A solitary osteolytic defect is seen roentgenographically, with no evidence of disease in the rest of the skeleton. The three year survival rate in this group was 32 per cent. The focus of plasma cell infiltration may remain localized for long periods of time. Eventually, multiple foci appear elsewhere in the skeleton. In this latter stage it is indistinguishable from multiple myeloma.

Multiple Myeloma (119 cases). The growth of multiple small intramedullary tumors results in disseminated osteolytic defects in bone, with the spine, skull, ribs and pelvis being most often affected. Anemia is more common in this group than in patients with the solitary variety. Myelomatous infiltration was reported in bone marrow studies of all but 8 of 96 patients thus examined. The three year survival rate was 5.3 per cent. No case survived for five years. Bronchopneumonia, severe anemia, uremia and cardiac failure were the most frequent terminal events.

Diffuse Myelomatosis (17 cases). A diffuse infiltration of the bone marrow occurs without discrete tumor masses. Diffuse osteoporosis may be seen roentgenographically but there are no diagnostic roentgen changes. Gradual onset of anemia is the usual reason for examining these patients. Once

severe anemia is manifest these patients deteriorate fairly rapidly, without remission, and are unaffected by treatment. The course of the disease is like that in severe aplastic anemia and the patients often succumb to pulmonary infections.

Extramedullary Plasmacytoma (6 cases). This is the most rare form of myelomatosis. The first manifestation is soft tissue tumor formation, which may later develop bony involvement.

The laboratory findings in these conditions are analyzed. Although an elevated erythrocyte sedimentation rate was present in 85 per cent, the rate was normal in the remaining 15 per cent. High readings were most common in the generalized form. Hyperglobulinemia was present in 70 per cent of the 118 cases tested and this was a result of an increase in the gamma component. Bence Jones protein was found in 35 per cent. The anemia is almost always normochromic and normocytic. There is a tendency to rouleau formation. Increased plasma cells are rarely seen. In general, an examination of the peripheral blood smear is of little diagnostic value.—*David Morse, M.D.*

Unander-Scharin, Lars. Melorheostosis; report of a case. *Acta orthop. scandinav.*, 1960, *30*, 101–106. (From: The Orthopaedic Clinic, Harnosand, Sweden.)

Melorheostosis is a very rare disease of bone first described by Leri and Joanny in 1922. It is characterized by continuous aching pain and limitation of motion, usually in one lower extremity. Presumably, the disease begins prior to birth or in early childhood and progresses very slowly. It may be associated with scleroderma or other soft tissue disturbance.

The roentgen features have been compared to liquid drops of wax which run down the long bones and are localized along the vessels and nerve paths. In small bones the changes are streaky or resemble ink blots. They have a "marblebone" appearance, but osteopetrosis can be differentiated because of its generalized nature.

The author describes a typical case of melorheostosis, which was followed for sixteen years. It was limited to the knee, lower leg, and foot of a patient who eventually underwent surgery for removal of a large mushroom-like exostosis from the medial femoral condyle.—*Arch H. Hall, M.D.*

Naide, Meyer, and Schnall, Charles. Bone changes in necrosis in diabetes mellitus; differentiation of neuropathic from ischemic necrosis. *A.M.A. Arch. Int. Med.*, Mar., 1961, *107*, 380–383. (From: Department of Medicine, Woman's Medical College of Pennsylvania and the Department of Medicine, Albert Einstein Medical Center, Philadelphia, Pa.)

The roentgen changes of osseous structures are of prime importance in differentiating ischemic from neuropathic lesions in the feet of diabetic patients. This differentiation is important because the treatment varies. In the neuropathic lesion, the treatment can often be limited to a toe amputation or incision and drainage, while in an ischemic lesion a leg or midthigh amputation is often necessary. Rarefaction, lysis or destruction of bone in a diabetic foot is often indicative of neuropathy and evidence of adequate blood supply, primary or collateral. On the contrary, in ischemic necrosis the circulation is impaired; therefore, no catabolism takes place and no bony changes occur.

There is a better prognosis in a diabetic patient with a soft tissue lesion of a foot if there is roentgen evidence of bone changes, indicating neuropathy, than if ischemic tissue necrosis is present and the bone is intact.

Two cases of diabetic patients were presented to illustrate the types of necrosis. The first patient had extensive bone destruction at the metatarsophalangeal joints and soft tissue necrosis secondary to diabetic neuropathy. Treatment was debridement. The lesion healed in six weeks indicating that the bone changes were neuropathic and not due to osteomyelitis. The second patient had the usual soft tissue ischemic necrosis typical of diabetes with arteriosclerosis but without roentgen evidence of bone changes. Because of severe pain and extension of necrosis on the foot, amputation at the midthigh was required.—*Jose Amorocho, M.D.*

Hansen, H.-J., and Ullberg, S. Uptake of S^{35} in the intervertebral discs after injection of S^{35}-sulphate. An autoradiographic study. *Acta orthop. scandinav.*, 1960, *30*, 84–90. (From: The State Veterinary Institute and the Department of Pharmacology, Royal Veterinary College, Stockholm, Sweden.)

Radiosulphate (S^{35}) was injected intraperitoneally into newborn piglets which were sacrificed at intervals up to sixteen days. Autoradiographs of sagittal sections of the spine were obtained to secure data regarding metabolic alterations in different morphologic structures.

The S^{35} concentrated in the epiphyseal cartilage, the inner zone of the annulus fibrosus, and the periphery of the nucleus pulposus. As a probable expression of synthesis of the sulphomucopolysaccharides, the incorporation of S^{35} at these sites accords closely with inferences deduced from morphologic features. Tissue layers which form the periphery of the nucleus and the perinuclear portion of the annulus represent the "growth zone" of the disk where fibroid or chondroid metamorphosis of the nucleus takes place.—*Arch H. Hall, M.D.*

ANDRÉN, LARS, and WEHLIN, LENNART. Double-contrast arthrography of knee with horizontal roentgen ray beams. *Acta orthop. scandinav.*, 1960, *30*, 307–314. (From: Roentgendiagnostic Department of the University of Lund, Malmo Allmanna Sjukhus, Malmo, Sweden.)

By employing a slight variation in the technique of double contrast arthrography of the knee, the authors have been able to demonstrate 62 of a total of 66 surgical meniscus ruptures. No false positives were obtained.

The technique is as follows: (1) The direction of the plateau is marked on the skin under fluoroscopic control; (2) the joint is aspirated under local anesthesia and 10 cc. of aqueous contrast medium plus an adequate amount of air is injected until the joint feels distended; (3) the patient walks about the room and flexes his knee several times; (4) the patient is placed on his side with the meniscus to be examined uppermost, and a firm pad is placed under the distal end of the femur with a small sandbag on the ankle to separate the joint; (5) film cassettes are placed against the knee and a horizontal roentgen-ray beam is used with a long film focus distance; and (6) the leg is rotated 10 to 15 degrees between exposures to show all parts of the meniscus. Additional views may be made in standard projections with a vertical roentgen-ray beam.

This method requires neither traction apparatus nor the presence of an assistant, and the dangers inherent in the knee-stretching procedures are eliminated. If air is injected first, one seldom gets extravasation of contrast medium into surrounding tissues. The authors agree that the value of any procedure varies with the skill and experience of the examiner, but they have found this technique to be fast, easy, and highly accurate in their hands.—*Arch H. Hall, M.D.*

BLOOD AND LYMPH SYSTEM

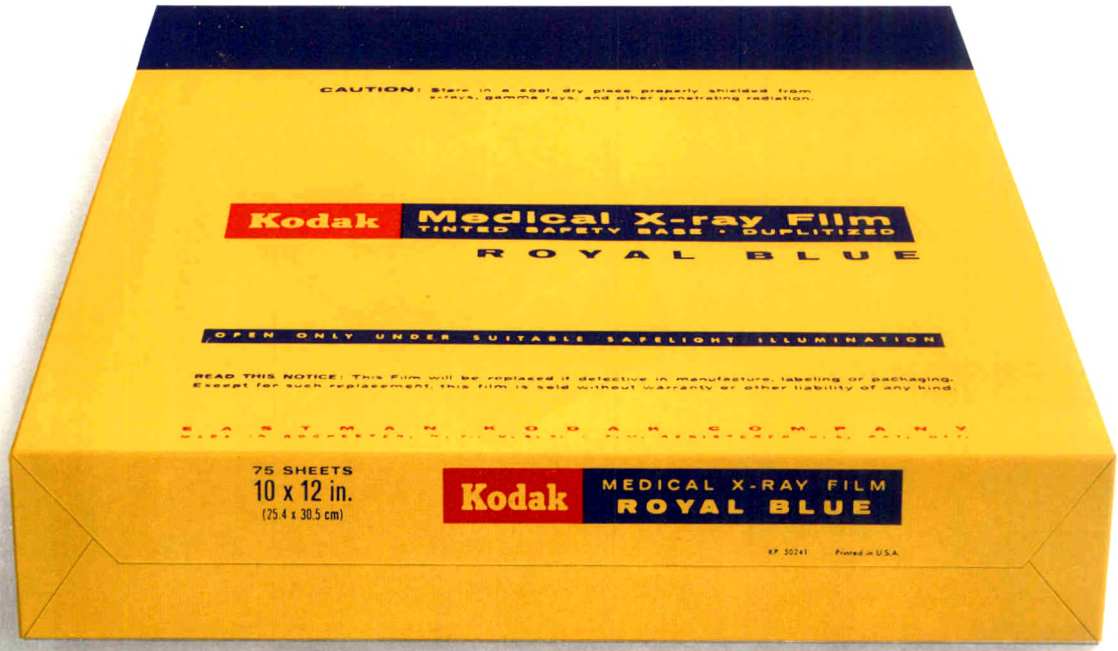
MONES, ROBERT. Vertebral angiography; an analysis of 106 cases. *Radiology*, Feb., 1961, *76*, 230–236. (Address: The Mount Sinai Hospital, Fifth Ave. and 100th St., New York 29, N. Y.)

The purpose of this report is to evaluate vertebral angiography as a diagnostic method in neurology and is based on 106 successful angiograms obtained in a two year period at the Mount Sinai Hospital in New York. A total of 114 attempts at direct vertebral angiography were made, 80 of which were successful. Inadvertently, 14 vertebral angiograms were obtained during the performance of routine carotid angiography and 12 during attempts at indirect filling in 37 patients. The indications for this examination were: subarachnoid hemorrhage; posterior cerebral artery disease; and either vascular disease or neoplasm of the posterior fossa. Fourteen transient complications were seen in the entire series of 128 patients with both successful and unsuccessful direct vertebral angiography. The 37 patients in whom indirect filling was attempted had no complications. Only one permanent complication occurred—an increase in the organic mental syndrome and visual field defect previously present.

Approximately one-third of the studies showed abnormalities. Of the 32 patients with a final diagnosis of basilar artery disease (proved or suspected), 16 had abnormal vertebral angiograms with 11 showing either narrowing or occlusion of a major artery and 5 showing some anomaly, such as an aneurysm or anomalous arteriovenous connection. Sixteen of the 30 patients with a final diagnosis of a tumor had abnormal angiograms. There were 22 posterior fossa tumors and 13 of these showed abnormal angiograms, while 3 of the 8 patients with supratentorial tumors had positive angiograms. While pneumoencephalography is the method of choice for diagnostic evaluation of posterior fossa tumors, incomplete filling or lack of filling of the ventricles may indicate the necessity for veretebral angiograms to attempt further localization. These angiograms may help by showing: (a) tumor stains, which give a good approximation of the position, size and, at times, the histology of the tumor; (b) deviation of an artery, sometimes indicating whether a tumor is intra- or extramedullary; and (c) the presence or absence of an aneurysm, which may act as a mass lesion.

The author feels that the amount of information gained from vertebral angiography is of value as a positive diagnostic test and as a means of ruling out with reasonable certainty certain vascular diseases, such as aneurysm, arteriovenous anomaly, or occlusive disease of the arteries. With care and experience these studies can be done, without undue risk, in patients suspected of having disease in the distribution of the posterior circulation.—*Donald N. Dysart, M.D.*

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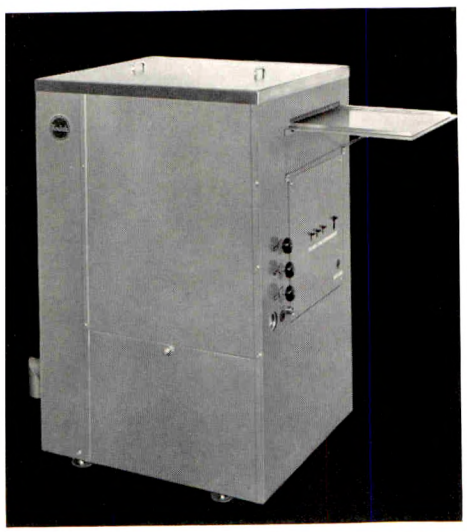
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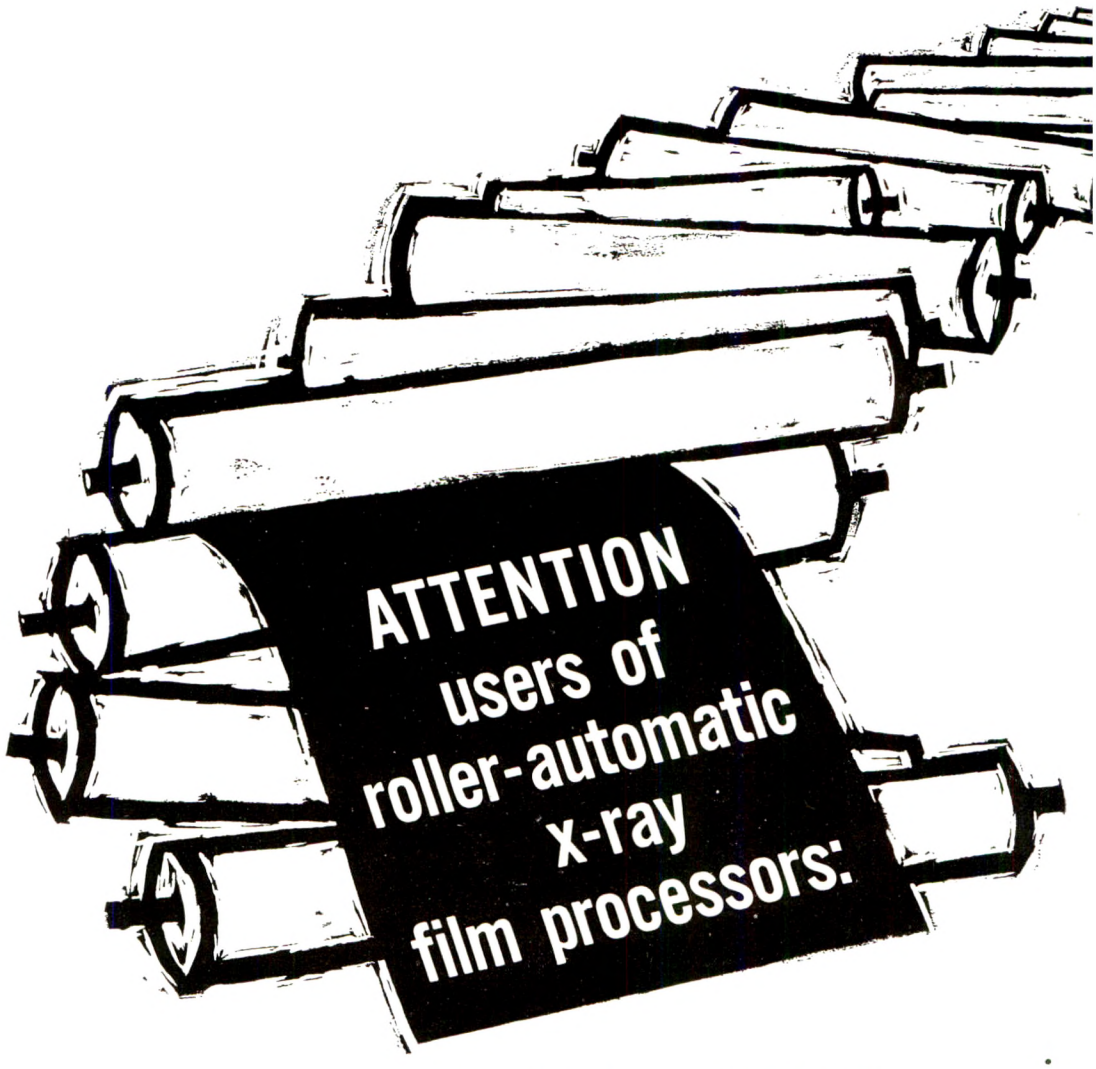
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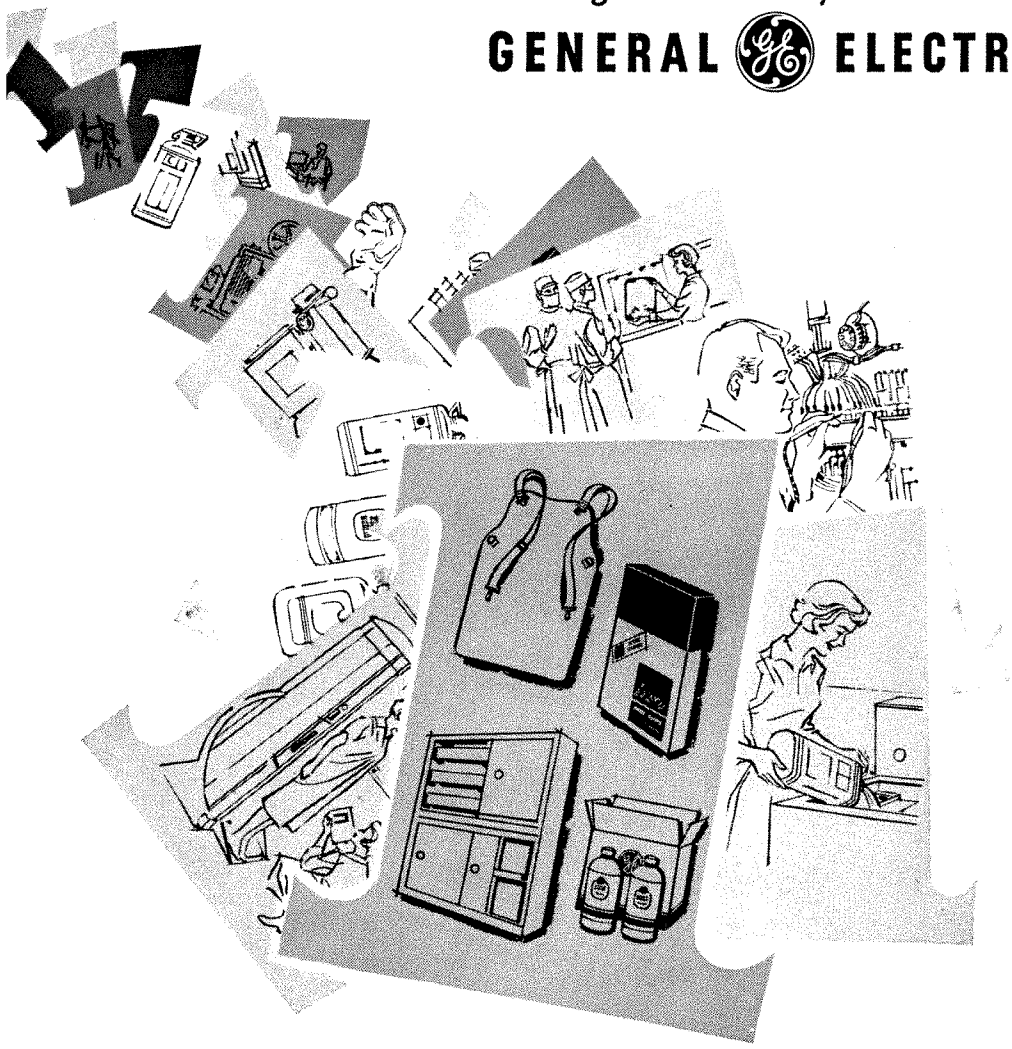


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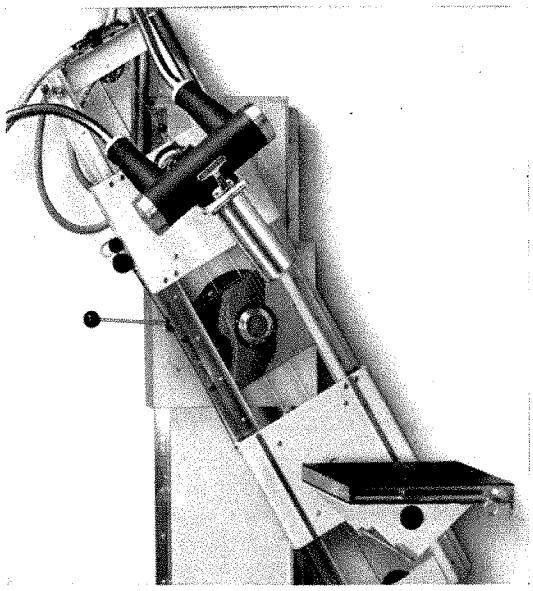
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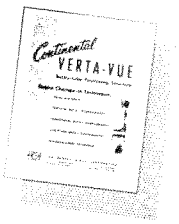
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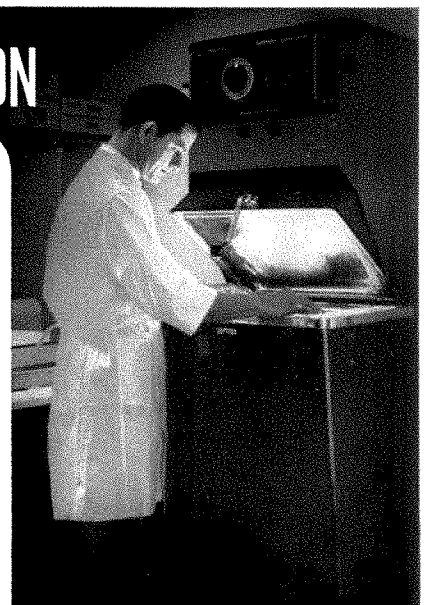
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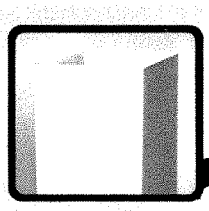
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1959. XVI, 379 pp., 168 ill. (some in colour) consisting of 301 details, cloth DM 65.–($16.25)

. . . The monograph is well balanced and excellently written; the author's short and clear sentences—one is only five words long—will be appreciated by foreign readers. The illustrations —mainly charts and line drawings—are numerous, generally simple in construction and help greatly in following the relevant text . . . About a thousand references, all with titles, are appended, and render the book valuable for consultation as well as for continuous reading. The book implements admirably the author's intention of providing a comprehensive monograph in this growing field of biology. It can be warmly recommended.

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Hartstrahltechnik

By Priv.-Doz. Dr. W. FRIK, Head of the Department of Roentgenology at the Clinic of Internal Medicine, University of Erlangen

1961. VIII, 144 pp., 76 ill. consisting of 100 details, bound in boards DM 28.50 ($7.15)

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By Doz. Dr. E. MUNTEAN, Graz

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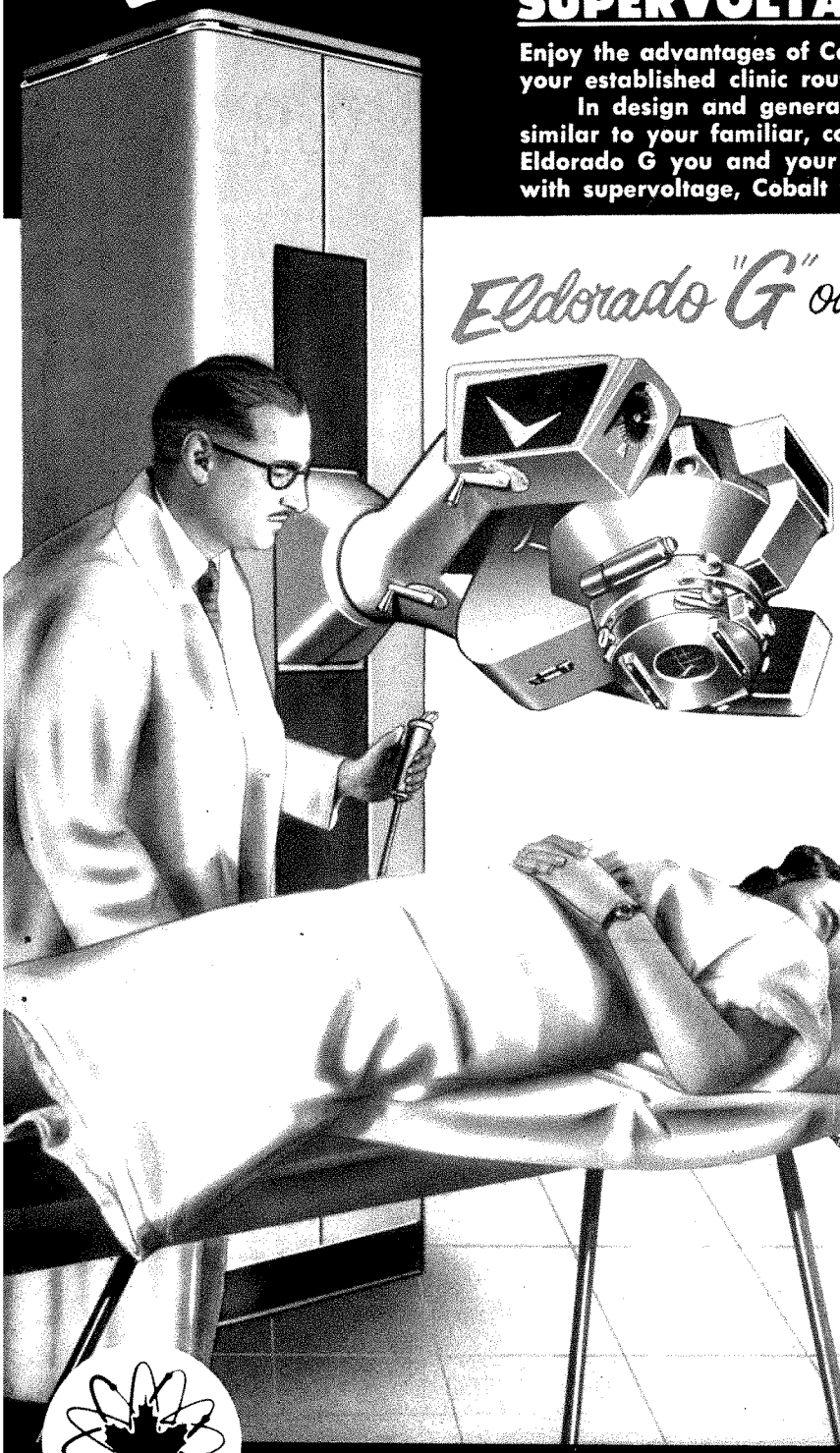
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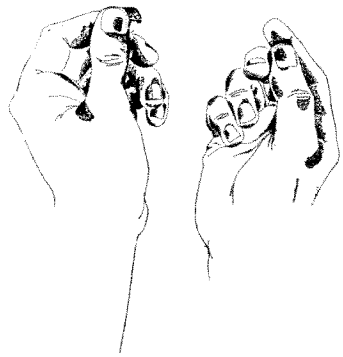
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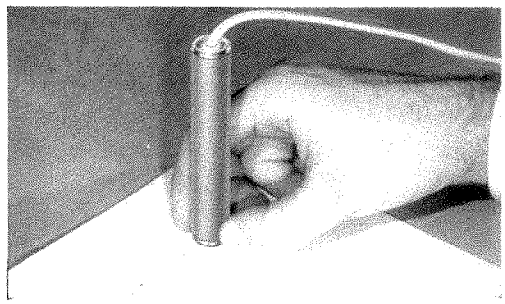


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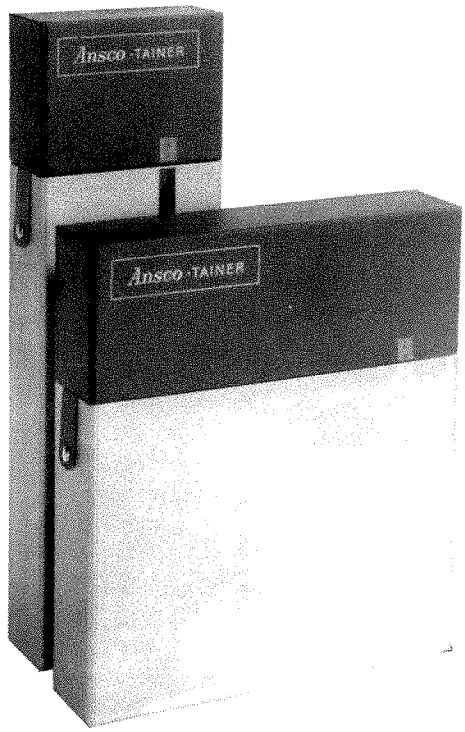
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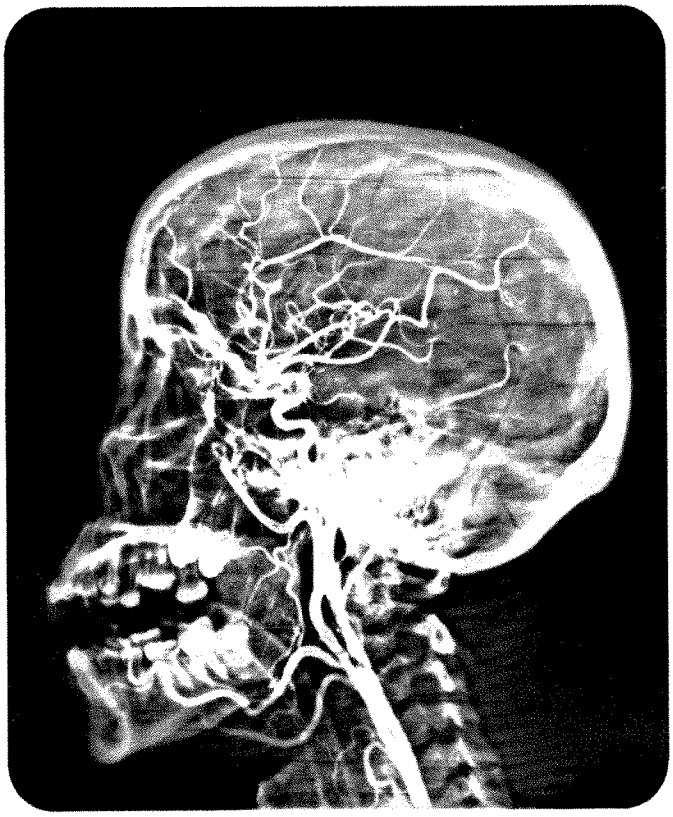
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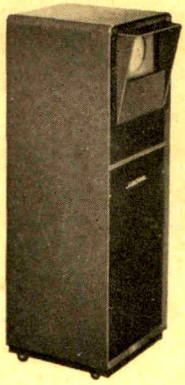
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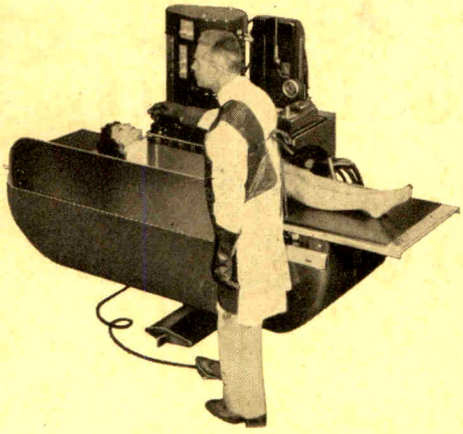
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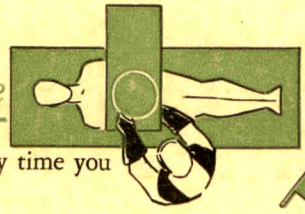
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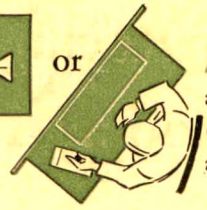


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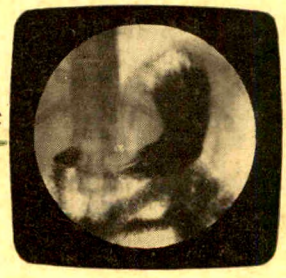


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